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THE
LARYNGOSCOPE.

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BRONCHOSCOPY IN ATELECTASIS IN THE NEWBORN.*

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Bronchoscopy in atelectasis in the newborn has now become an accepted endoscopic procedure. During the past few years, the otolaryngologist associated with a general hospital has been called upon not infrequently by the obstetrician and pediatrician to aid in the treatment of newborn infants with respiratory distress. During this time the results have clearly shown that laryngoscopy and bronchoscopy of newborn infants are procedures which can readily be undertaken by the careful, trained operator without serious harmful effects to the patient. The operation is of both diagnostic and therapeutic value.

General Considerations:

Atelectasis is defined in Dorland's American Illustrated Dictionary as either an "imperfect expansion of the lungs at birth" or as "partial collapse of the lung." The word derives from two Greek words meaning "imperfect expansion."

*Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1950.

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In medical literature there are four different conditions in the lung which are referred to as "atelectasis." The one most acceptable is fetal atelectasis, in which there is a failure of the lungs to expand at birth. The second type is the condition in which the lung is collapsed as a result of external pressure such as hydrothorax. The third is a condition known as "postoperative atelectasis"; the histopathologic aspects of which are inedaquately described because material is seldom available in the acute phase. The fourth is the condition in which the lung becomes airless and contracted after obstruction to the bronchus from an obstructive lesion, such as neoplasm.[1]

Physiology and Pathology:

Holt[2] states that in fetal atelectasis there is a persistence of the fetal state in the whole or in any part of the lung. The lung of the fetus is a uniform dark red color, and it shows lobular divisions very distinctly upon its surface. It is firm and solid, and sinks readily in water. Connective tissue is abundant and forms distinct fibrous septa which stretch across the lungs in every direction.

MacMahon[3] describes a separate pathological entity, congenital alveolar dysplasia. This is a morphologic anomaly of the lungs of newborn children. It is suggested that this anomaly represents a retardation and disturbance in the normal development of pulmonary alveoli. The etiology, at present, is obscure.

According to Brennemann,[4] in the intrauterine life the child's lungs are atelectatic. Twenty minutes after birth there is a 17 cc. capacity, and three to six hours after birth the capacity is 36 cc. Normally it is several days before the lungs have completely expanded. This expansion becomes complete first in the anterior borders and apices. The paravertebral, central and posterior portions are the slowest to expand. The inflation is sometimes irregular because of mucus in some of the bronchi.

Patterson and Farr[5] introduce strong evidence based on animal experimentation in support of the hypothesis that the

human fetal respiratory tract is not inert in utero, but is subject to rhythmic respiratory movements, during which there is a tidal flow of amniotic fluid through the bronchial tree and alveoli. Examinations of lung secretions of neonatal deaths, some stillborn, showed amniotic fluid present. That this is not entirely due to passage through the birth canal, as is generally thought, is shown by the fact that some of these cases were delivered by Caesarean section. Snyder and Rosenfeld[6] showed that India ink injected into the amniotic sac is followed shortly by the appearance of this material in the pulmonary alveoli of the animal fetus.

Wilson and Farber[7] feel lack of expansion in the premature is not a failure of respiratory effort in many instances, though this may be weak, but that it is due to cohesion of moist surfaces of the air passages. This condition may, of course, be emphasized by any disturbance in the respiratory center, imperfectly developed thoracic mechanism or thorough obstruction of bronchi by aspiration of amniotic fluid, mucus or blood.

Incidence:

In an attempt to determine the incidence of pathological fetal atelectasis the number of cases so diagnosed in a general hospital of 340 beds was studied (see Table 1). In a period

TABLE 1.

	Number of Births (Living)	Infants Deaths (Neonatal)	Still-born	Congenital Atelectasis
1944	1,700	39	34	4
1945	1,746	48	47	7
1946	2,100	55	55	5
1947	2,352	62	60	0
1948	2,202	33	57	6
Total	10,100	237	253	22

of five years (from 1944 to 1948, inclusive), it was found that out of 10,000 births there were 22 cases of fetal atelectasis, 15 of which expired and came to autopsy.

The postmortem findings are shown in Table 2. It is to be noted that in this series there were very few cases in which atelectasis *per se* was the primary cause of death. This is in accordance with the findings of Patterson and Farr,[5] who found in a series of 12 neonatal deaths that only five, or 42 per cent, were considered to have died from this condition. In the series here presented, six, or 40 per cent, expired from uncomplicated fetal atelectasis.

Ethel Dunham[8] states that "in cases of atelectasis in the newborn, failure of proper functioning of the respiratory center is generally held to be the most frequent cause of abnormal persistence of the fetal state in some parts of the lungs. Often the cause of the failure of the respiratory center cannot be determined, but immaturity, asphyxia, hemorrhage of the brain or edema may be responsible for the condition. Mechanical factors such as plugging of a bronchus by mucus or pressure on the lung by an enlarged heart or possibly by an enlarged thymus gland are said to have caused atelectasis of the lungs occasionally in the newborn infant. Atelectasis of the lungs in the newborn infant is therefore never primary but always secondary to some condition that interferes with the mechanism bringing about full expansion of the lungs."

Hemorrhage, edema, hydrocephalus, congenital malformations, bronchopneumonia, etc., were some of the contributing factors which appeared in this series. There was only one case in which variations in the Rh factor appeared.

It is noteworthy that in this group, eight, or 53 per cent, were prematures. Two were delivered by Caesarean section; and three were breech presentations.

The duration of life varied from approximately one minute to eight days.

Signs and Symptoms:

The classical picture of newborn atelectasis secondary to bronchial obstruction is, according to Owens:[9]

TABLE 2.

Case No.	Period of Gestation	Method of Delivery	Initial Respiratory Movements	Duration of Life	Gross Appearance of Lungs	Anatomical Diagnosis
(1)	Nine months	Breech difficult	Weak difficult resuscitation	Eight days	Expanded	Bilateral patchy atelectasis Incomplete development of adrenals Impetigo Mongolian (?) Hemorrhage and thrombosis of choroid in right lateral cerebral ventricle
(2)	Seven months	Spontaneous vertex	Weak	Twenty-three hours	Partial collapse	Prematurity Partial primary atelectasis Bronchopneumonia Neural thrombosis l. ventricle Subdural hematoma Extramedullary hematopotises
(3)	Seven months	Breech difficult	Weak	Twenty-four hours	Partial collapse	Prematurity Congenital atelectasis, bilateral
(4)	Six months	Vertex spontaneous	Weak	Twenty minutes	Massive collapse	Prematurity Atelectasis Hemaperitoneum Hematocoele Petechial hemorrhage in subarachnoid
(5)	Seven months	Vertex spontaneous	Weak: baby did not breathe for 10 minutes	Twenty hours	Partial collapse	Prematurity Cerebral edema Hydrocephalus Atelectasis
(6)	Eight months	Caesarean section	Weak	Thirty minutes	Massive collapse	Prematurity Congenital atelectasis (Mother Rh negative, Father Rh positive)

TABLE 2 (Cont.).

Case No.	Period of Gestation	Method of Delivery	Initial Respiratory Movements	Duration of Life	Gross Appearance of Lungs	Anatomical Diagnosis
(7)	Eight months	Spontaneous vertex	Weak	Twenty-four hours	Massive collapse	Atelectasis left lung and right lower lobe
(8)	Nine months	Low forceps vertex	Weak	One hour	Massive collapse	Atelectasis Congenital malformation of right diaphragm Diaphragmatic hernia (herniation: liver, small intestine, ascending colon and cecum through malformation)
(9)	Nine months	Caesarean section	Weak	Twenty-four hours	Massive collapse	Atelectasis left lung A subarachnoid hemorrhage
(10)	Seven months	Breech	Weak	Twenty-four hours	Massive collapse	Prematurity Atelectasis
(11)	Nine months	Spontaneous vertex	Weak	Twenty-four hours	Partial collapse	Bilateral pulmonary atelectasis Bilateral subarachnoid hemorrhage Dilated left ureter
(12)	Nine months	Spontaneous vertex	Weak	One hour	Massive collapse	Atelectasis, partial on right, complete on left Double kidney pelvis Anomalous ureters bilateral Congestion cerebral vessels
(13)	Seven months	Spontaneous vertex	Weak	Three hours	Massive collapse	Atelectasis Bronchopneumonia Neural thrombosis Subdural hematoma
(14)	Nine months	Vertex precipitous	Weak	Thirty minutes	Massive collapse	Congenital atelectasis
(15)	Nine months	Vertex spontaneous	Weak: two breaths only	One minute	Massive collapse	Congenital atelectasis

1. Progressive dyspnea with cyanosis, most marked after crying or other effort. This may often be temporarily relieved with oxygen. When these symptoms are not present, listlessness and pallor are usually noticed.

2. Suprasternal retraction with diaphragmatic tug on the lower ribs and diminished thoracic expansion on one or both sides, associated with suppressed breath sounds, with or without percussion dullness.

3. Coarse inspiratory rales and areas of localized emphysema. Coarse moist rales are an important finding and are usually absent in cases not due to body secretions.

4. Dehydration.

5. X-ray of the chest will usually show a rather complete atelectasis of one or more lobes. An X-ray should be made when possible in every case, but the diagnosis is made primarily on the clinical picture. The chest X-ray is also most helpful in distinguishing pulmonary from nonpulmonary entities.

Treatment:

The first requisite in resuscitation of the apneic newborn is the establishment of a free airway. Conservative measures such as catheter suction and external stimulation should first be tried. The major point to be emphasized with such stimulation is gentleness: the risk of causing visceral trauma, increased shock, and cerebral hemorrhage is very real. Oxygen is to be administered and warmth maintained.

Snyder,[10] in discussing the usefulness of respiratory stimulants such as alpholobeline, coramine, caffeine, metrazol and cyanide states that "it has been difficult to evaluate various reports concerning their rôle at the time of birth since these drugs are administered usually following labor, which is complicated by various factors that may alter respiratory activity, such as anorexia, anesthesia and trauma." In order to determine the effect of these respiratory stimulants upon the normal newborn, Lim and Snyder[11] carried out experi-

ments on newborn rabbits immediately following delivery. Results showed that response by increase in rate or depth of breathing was brief, lasting less than a minute; furthermore, death frequently followed the onset of convulsions resulting from the administration of alphalobeline, caffeine or coramine.

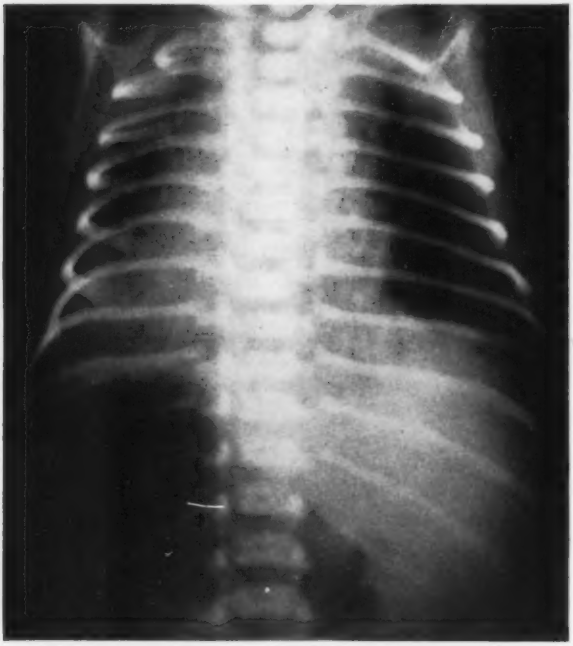

Fig. 1. X-rays from Case 1 showing fairly good ventilation in the lung fields with a wide heart shadow.

It has been the custom in some institutions to give vitamin K more or less empirically in cases of this kind in an attempt to control or modify hemorrhage. Fluids are to be given to control dehydration; and chemotherapy should be used to minimize the infection which usually accompanies prolonged atelectasis.

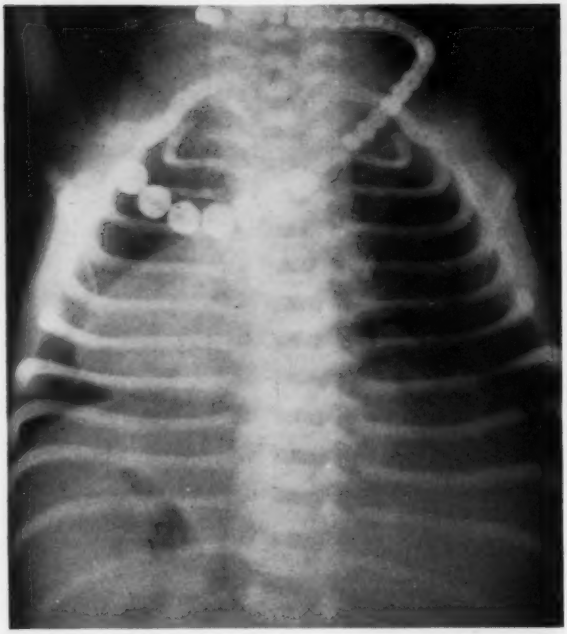

Fig. 2. X-rays of Case 2 with small area of atelectasis involving the posterior portion of the right lobe, with no shift of the central shadows.

Indications for Bronchoscopic Intervention:

Because of the great variations in the clinical picture, and rapid changes in the condition of atelectatic infants, final decision as to need for further treatment should rest with the bronchoscopist and pediatrician. The indication for bronchoscopic intervention, according to Owens,[9] is "any condition in which there is mechanical obstruction of the trachea or bronchus, not correcting itself spontaneously or with the use of approved conservative therapy, and which appears to be leading to the infant's exhaustion."

Following successful bronchoscopic aspiration, there is usually a progressive improvement in the infant's respiration; however, complete relief most frequently occurs six to eight hours after instrumentation.

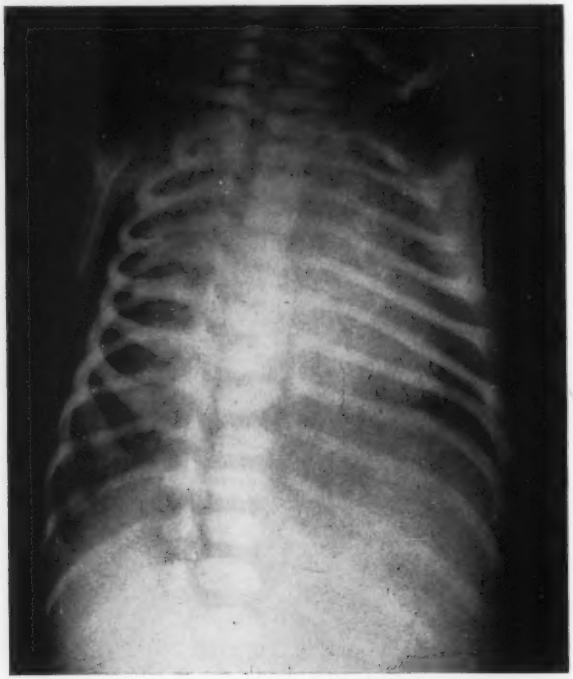

Fig. 3. X-rays from Case 3 showing bilateral atelectasis with very little ventilation on either side.

Case Reports:

The following six cases diagnosed as congenital atelectasis of the newborn have been bronchoscoped during the past eight months:

Case 1: Diffuse congenital atelectasis, from bronchial obstruction by body secretions.

A five-pound, one-ounce boy was born by Caesarean section. Because of dyspnea and cyanosis, it was placed in an incubator with an attached oxygen unit. Cyanosis appeared each time it was removed from the incubator. X-ray showed some fair ventilation of the lung fields, but the cyanosis and dyspnea persisted into the second day despite conservative treatment.

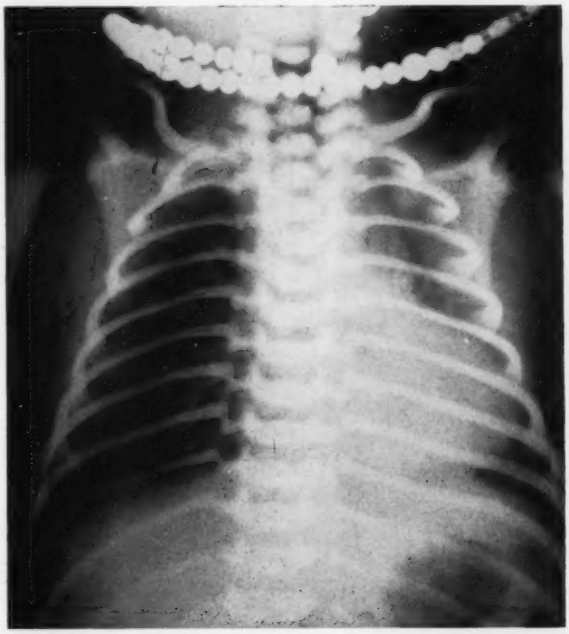

Fig. 4. X-rays showing massive atelectasis of the left lung with shift of the central shadows.

There being no improvement on the third day, an infant bronchoscope was passed. No marked obstruction was noted but a moderate amount of mucoid material was removed by suction. The entire procedure lasted approximately three minutes.

Symptomatic improvement was prompt and the child was discharged on the tenth day.

Case 2: Congenital atelectasis of the right lower lobe.

A six-pound, eleven-ounce baby boy, L. O. A., with episiotomy and low forceps, showed cyanosis and dyspnea with tugging. Placed in an incubator with attached oxygen unit, the cyanosis reappeared whenever the child was removed from the unit.

An X-ray of the chest showed a small area of atelectasis involving the posterior portion of the right lower lobe. There was no shift of the central shadows.

An infant bronchoscope was passed and suction applied to the right main bronchus. The entire procedure was completed in less than three minutes.

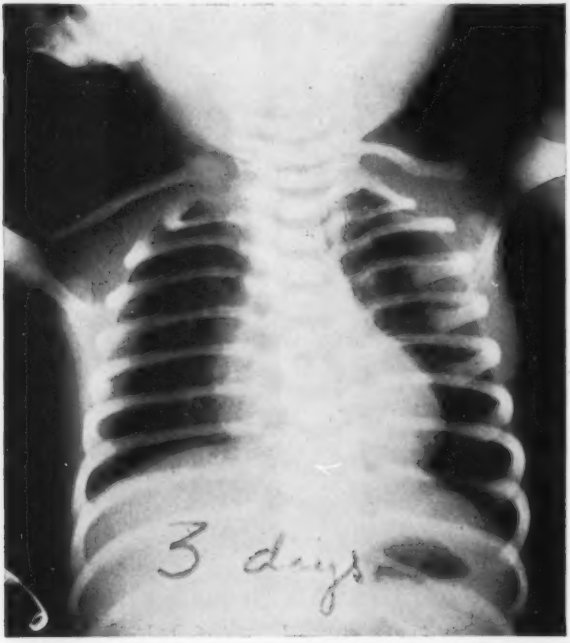

Fig. 5. X-ray showing appearance of chest three days after bronchoscopy.

Following bronchoscopy, the child's cry improved and the periods of cyanosis became less frequent. He was discharged on the tenth day.

Case 3: Bilateral massive atelectasis with bronchopneumonia.

A six pound, two and one-fourth-ounce boy, R. O. A., low forceps, developed rather marked cyanosis and dyspnea the second day. There was no improvement with mouth suction and frequent stimulation.

The child was placed in an incubator with attached oxygen unit, but little marked improvement was noted. An X-ray showed bilateral atelectasis with very little ventilation on either side. The heart shadow was large and indistinctly seen.

Two days following the onset of cyanosis and dyspnea, the child was bronchoscoped, conservative therapy having proved to no avail. Suction was applied and a moderate amount of mucus removed. No gross obstruction was seen.

There was no marked improvement after the bronchoscopic procedure, and the child expired 14 hours later. Unfortunately, no autopsy was obtained.

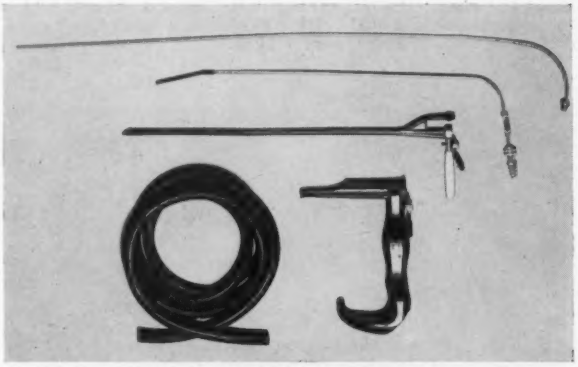

Fig. 6. Instruments used in bronchoscopic aspiration for atelectasis in the newborn. From above; infant suction tip, Samson infant aspirator, newborn bronchoscope, rubber suction tubing and newborn laryngoscope.

It was felt at the time that there was undoubtedly an accompanying central lesion, probably hemorrhage in this case; however, bronchoscopy was attempted without any apparent beneficial result.

Case 4: Fetal atelectasis, massive.

A seven pound, seven and one-half-ounce baby boy, L. O. A., born spontaneously, responded poorly to conservative methods of resuscitation, was bronchoscoped within two hours of birth. Large amounts of thick mucus were removed by suction.

Following bronchoscopy, respirations continued rapid and labored, but the color showed marked improvement. It was also noted in the physical examination that there was a supernumerary left ear.

The following day dyspnea and cyanosis were still marked and there was considerable tugging. Bronchoscopic aspiration was again helpful in somewhat relieving the cyanosis. At this time it was noted that the larynx showed no evidence of trauma from the previous manipulation.

Despite this second bronchoscopic drainage, the cyanosis returned and the dyspnea became more severe. The child expired from exhaustion approximately 14 hours after the second bronchoscopic procedure.

Postmortem examination showed both lungs smooth, glistening and maroon colored with no crepitation. It was of the consistency of liver. The bronchi contained a considerable amount of mucoid material.

Microscopically, all sections showed massive atelectasis with acute and subacute areas of pneumonitis with terminal bronchioles containing evidence of vernix caseosa.

Case 5: Congenital atelectasis, massive.

This male infant, weighing three pounds, fourteen ounces, was born by spontaneous delivery. Although premature, he was resuscitated without difficulty, and his color was good. Breath sounds were heard bilaterally throughout the chest; the only abnormality being a superficial hemangioma of the forehead and scalp. He was put in a heated crib, and given a Synkavite series and continuous oxygen.

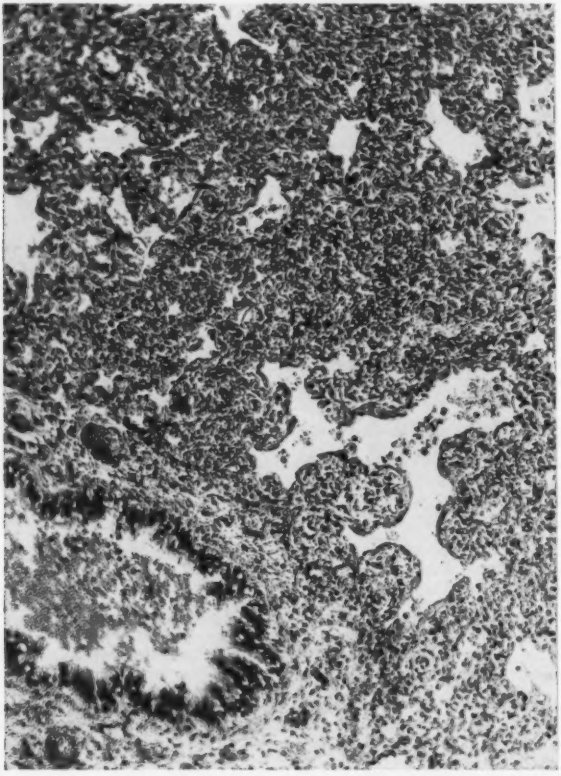

Fig. 7. Photomicrograph of atelectatic lung showing bronchiole and alveoli filled with aspirated amniotic contents and bronchial debris.

Two days later, at 8:00 A.M., the child's color was poor, lips cyanotic and respirations labored. The formula was gavaged at 10:00 A.M. His color continued poor, respirations became more labored, and since chest retractions were noticed, bronchoscopy was decided upon.

A moderate amount of mucoid material was withdrawn by bronchial suction. There was a minimal amount of trauma to the larynx and the patient's postoperative condition was good.

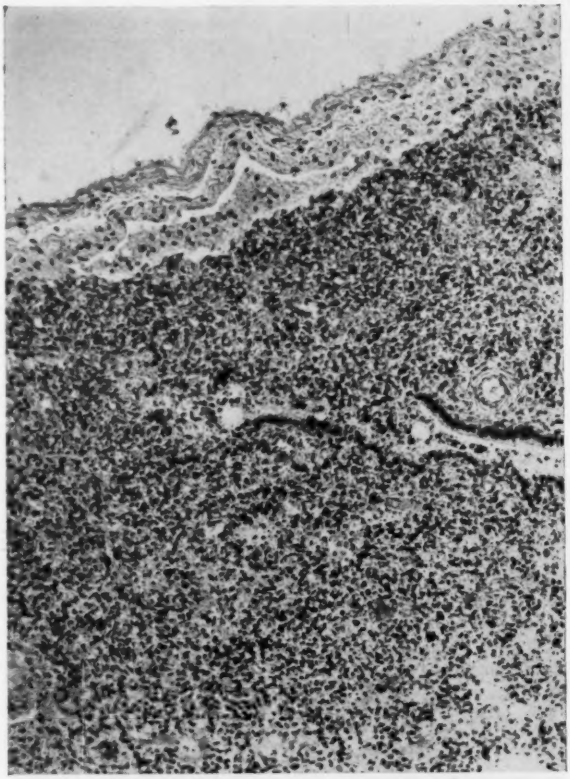

Fig. 8. Photomicrograph of atelectatic infant lung (pleura above).

At 5:00 P.M., the child was crying lustily and respirations were deeper. There was no chest retraction, and color was good. The following day the child showed marked improvement and no further bronchoscopic aspiration was necessary.

Comment:

Six cases of atelectasis in the newborn in which bronchoscopic aspiration was used as an adjunct to treatment have been presented. Two of the cases had a fatal outcome; however, in neither was there any evidence of harmful effect caused by the bronchoscopic procedure.

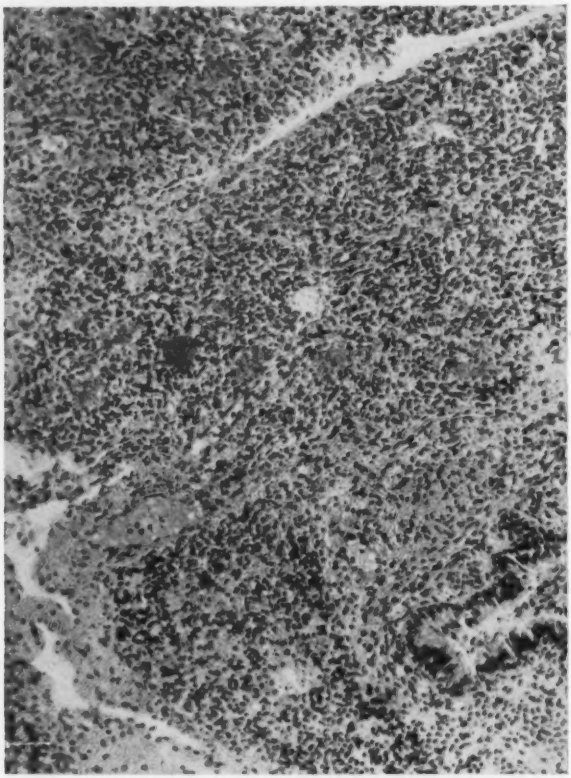

Fig. 9. Photomicrograph of normal expanded infant lung.

Very little equipment was necessary. The instruments used were the Holinger newborn bronchoscope, the Holinger new-

born laryngoscope, the infant suction tip and the Samson infant aspirator. These are readily portable and may be moved from one hospital to another.

In each case an oxygen unit and sterile tracheotomy set were available for immediate use. Although extreme care was used in all cases in approaching and entering the glottis, in no instance did the entire endoscopic procedure consume more than three and one-half minutes. In two cases it was

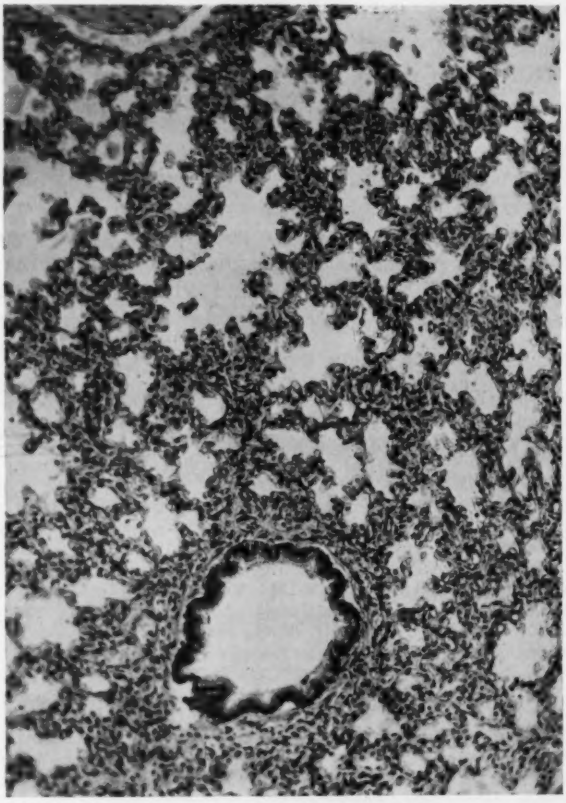

Fig. 10. Photomicrograph showing atelectasis and pneumonitis.

deemed necessary to give artificial respiration for several seconds before spontaneous respiration began.

It is most important to bronchoscope the infant before pneumonitis has a chance to develop in the atelectatic lung. Moersch[12] and others feel that such areas may perhaps be precursors of bronchiectasis.

Summary and Conclusions:

1. Bronchoscopic aspiration of the newborn is a relatively benign procedure when properly performed.

2. Selected cases of newborn atelectasis, secondary to bronchial obstruction, which failed to respond to conservative treatment, are materially benefited by bronchoscopic aspiration.

3. The etiology of atelectasis in the newborn is still not fully understood.

4. If bronchoscopic aspiration were more widely used for all types of congenital atelectasis, the indications and contraindications would be sooner understood and more lives undoubtedly saved.

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THE ROLE OF BACTERIAL INFECTION IN RESPIRATORY ALLERGY.*

LOUIS E. SILCOX, M.D.,
Philadelphia, Pa.

The otolaryngologist is so frequently confronted with the difficult problem of satisfactory treatment in cases of chronic sinusitis that an investigation into one of the important factors in its pathogenesis seems warranted. Most observers agree that between 70 and 80 per cent of all cases of chronic sinusitis have allergy as their basic pathology, and without adequate attention to this factor the treatment of this common disease process often ends in failure. The atopic or non-bacterial allergies have been exhaustively studied in reference to the respiratory tract,[1] but there is scant fundamental clinical data on bacterial sensitivity and the rôle it plays in the pathogenesis of chronic rhinitis, sinusitis and asthmatic bronchitis.[2-5]

Many clinicians are unwilling to accept hypersensitivity to infecting bacteria as an explanation of these conditions because the skin reactions obtained with bacterial products are of a delayed and inflammatory nature instead of the immediate urticarial type obtained with simple protein extracts. Some[6] believe that individuals possessing foci of infection, either in tonsils, teeth, sinuses or prostate, acquire a sensitiveness to bacterial protein which is similar to that of non-bacterial allergens, and that the infecting bacteria and their products alone and without the intervention of other foreign proteins may result in allergic symptoms. Billings,[7] over 30 years ago, was cognizant of focal infection as a cause of

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anaphylaxis. He stated that the bacterial protein of the pathogenic micro-organisms of the focus may sensitize the body cells.

When a foreign protein—bacterial or nonbacterial—finds its way parenterally into the animal body certain changes take place in the body as a result of the contact with that protein. The exact nature of these changes is obscure, but the effects which they may produce if the same protein reaches the tissues again are quite familiar as the various hypersensitive reactions to foreign proteins. The reaction may come in the form of anaphylactic shock, asthma, allergic rhinitis, hay fever, serum sickness, etc.[8]

Zinsser[9,10] has pointed out that in allergy to bacterial infection we are dealing with the sensitization of the body by autolytically liberated antigenic substances which are absorbed from any focus in which bacteria react with inflammatory tissues, and as a result of which the body is subsequently rendered sensitive to contact with these same autolytic products, whether they are liberated and absorbed from a chronically existent focus or from an identical infection subsequently acquired.

Allergy to bacteria or their products is a phenomenon connected with many diseases such as tuberculosis, streptococcal infection, undulant fever, typhoid fever, etc., but best known in tuberculosis because of the discovery of the allergic phenomena in the skin of those who were infected by the tubercle bacillus.

That allergy takes part in the specific defense of the organism against a variety of substances, both bacterial and nonbacterial, is definite.[11] In tuberculosis after the body cells have once been sensitized by previous infection with tubercle bacilli allergy of varying degree, from a slight hyperemia to a severe exudation, may be met with whenever reinoculations of bacilli or bacillary protein takes place—the degree of allergy depending upon the dosage and virulence of the bacilli and the reacting capacities of the host. Such allergy is a specifically acquired reaction and is usually considered as part of the host's protective mechanism.

Two different types of immunologic reactivity were demonstrated by Zinsser.[9] Both the anaphylactic and the tuberculin types of sensitization were produced in the same animal. The "anaphylactic" type of reaction appeared in from two to 15 minutes after intradermal injection of the antigen. It expressed itself in the development of a growing wheal often surrounded by an areola. This reaction may last from one-half hour to one or two hours and fades without leaving any trace of injury to the tissue. The "tuberculin" type of skin reaction showed no immediate effect, but within four or five hours a swelling becomes apparent and in the course of 12 to 24 hours there is a swollen edematous area of varying intensity, often with a central necrotic spot and occasional hemorrhage. This reaction may not reach the height of development until 48 hours after the injection, and is accompanied by distinct signs of inflammation and some cell death.

The anaphylactic response on the part of the body to bacteria or their products is comparable in all respects to the nonbacterial sensitization, and the tuberculin type allergic response may be caused by any bacterial organism. Some inflammatory reactions of the body are necessary for its formation. Circulating antibodies are not a significant feature in the tuberculin type but definitely are in the anaphylactic type.[12]

If an animal is immunized with whole bacteria—either living, formalinized or killed in some other manner that avoids dissolution of the cell substance—we obtain antibodies which are type specific and bring about the well known test-tube reactions of agglutination, precipitation, opsonization, etc. If the bacteria are subjected to even the most gentle chemical cleavage the bacterial antigen may be split into at least two antigenically functioning fractions: one, a nitrogenous nucleoprotein-like material, the other a partial antigen, probably of carbohydrate structure which has been called the "soluble specific substance" by Avery.[13]

Bacterial allergy may be defined as a condition in which the body is sensitized to a bacterial antigen and this antigen in its most potent form results from biological disintegration of

the bacteria, either by autolysis or similar disintegration which takes place in inflammatory foci; and it is likely that many of the experimental difficulties that have been encountered are due to the diminution of functional properties coincident to chemical manipulation *in vitro*. A formidable obstacle in explaining the specificity of bacterial effects lies in the extreme complexity of bacterial antigens and antibodies as compared with those of the simpler coagulable proteins.

The allergic state represents an increased capacity on the part of the tissues to react to this antigen, which may either come to the body in the form of a new infection or through the lighting-up of an old focus. This increased capacity to react may be injurious and result in pathological changes and disease.

The Arthus phenomenon is a special type of accelerated allergic response in rabbits and other animals. The Schwartzman phenomenon is an unexplained skin response to bacterial filtrates. These reactions are effects coming from bacterial metabolism and the corresponding complexity of reactions on the part of the host. It is entirely conceivable that some of these specific and nonspecific bacterial agencies enact an appreciable part in the pathogenesis of hyperplastic sinusitis, infectious asthma and other hypersensitive clinical states attributable to bacteria. It is evident that there are several types of allergy which differ particularly in the location of the antibody. In protein anaphylaxis the antibodies are in the cells in large numbers and are similar to those present in the sera. Their location and concentration are the factors which determine the suddenness of the reaction. In bacterial allergy the antibodies are also in the cells, but they arise from the activity of the infectious process and depend upon a third factor. In immunity they are almost exclusively in the circulating blood. Skin reactions are small, the resistance to infection is high and their concentration in the serum is the most important factor.

Whenever we find allergic phenomena in the course of bacterial disease we must assume that the reaction is due to some substance or substances produced during the growth of

the causative micro-organism or derived from their intimate contact with the patient's cells after they have been acted upon and sensitized by a previous identical infection. The result of this contact is an inflammatory response.

There is no doubt but that bacterial infection of the respiratory tract may cause hyperplastic sinusitis and asthma. The latter may be acute paroxysmal or the chronic form. There have been some doubts as to whether hyperplastic sinusitis and asthma of infective origin can be considered as allergic reactions. This problem has been reviewed by many students of the subject and controversy still exists.

Some of the evidence in favor of an allergic response to bacteria or their products is the presence of an increase in the number of eosinophiles locally and in the circulating blood. Cooke[14] has reported an eosinophilic infiltration in hyperplastic rhinitis and sinusitis, and in a series of 131 active cases of asthma he found that a blood eosinophilia is not only more frequent but it is also on the average higher in infective asthma than in atopic asthma. He feels that eosinophilia of the tissues and secretions of sinuses and bronchi is an allergic reaction and cannot be due to the bacterial infections alone. Campbell[15] states that eosinophilia represents a cellular response which invariably accompanies local anaphylaxis and may or may not be evident in the general circulation, depending upon the severity of the hypersensitive reaction. Substances, such as adrenalin, which diminish or prevent anaphylaxis also diminish or prevent eosinophilia.

That the eosinophiles of the sputum in asthma and antral exudates in allergic sinusitis are probably of local tissue origin and not myelogenous is indicated by the fact that 15 cases with normal differential counts of the peripheral blood showed abundant eosinophiles in the secretion or exudate of bronchial and nasal mucous membranes and careful study of smears showed that the eosinophiles of the exudates are not all morphologicaly similar to those of the blood taken at the same time. Hyperplastic sinusitis and infective asthma may be re-

garded as an allergic reaction quite as properly as is the skin sensitive type—although the immunologic reactions in the two forms are not identical.

It is a well known fact that bacteria may be present in the sinuses or sinus mucosa in the absence of apparent infection.[16] The streptococcus is not a pus producing organism but tends to create edema and carry on in a subacute or chronic form. Virulent streptococci have been cultured from nonsuppurating hyperplastic sinusitis. Schenck[17] makes the observation that allergic ethmoidal tissue cannot combat infection effectively, and chronic infection results which favors bacterial sensitivity.

Cooke[18] states that almost 100 per cent of the pathologic manifestations of sinusitis are of the hyperplastic or thickened membrane type. Eighty per cent of the membranes he cultured were positive for one or more organisms.

A true hyperplastic membrane is much thickened, often infolding and polypoid with desquamation of the cilia, a much thickened hyalinized basement membrane and in the tunica propria marked edema, glandular hyperplasia, proliferation of the connective tissue and eosinophilic, round, and plasma cell infiltration. In the chronic cases neutrophiles are not frequent.

Grove and Cooke[18] felt that hyperplastic sinusitis is caused solely by infection which produced a special pathologic response conditioned by the allergic constitution of the patient. Grove and Farrior[2] found bacteria in 80 per cent of the ethmoid and sphenoid membranes at operation, and 96 per cent of the maxillary sinus membranes showed bacterial growth. The staphylococcus was the predominate organism and the hemolytic streptococcus was found twice as often in the antral membranes as in the ethmoid and sphenoid membranes. These allergic patients all had chronic hyperplastic sinusitis.

A comparison of organisms obtained from 87 maxillary sinus membranes with those grown from previous washings of the antra showed 43.7 per cent were different from any recovered preoperatively. Stained sections of tissues removed

from the sinuses demonstrated bacteria very definitely in 85 per cent. The bacteria recovered from 108 antral membrane cultures were as follows:

Staphylococcus	57%
Streptococcus viridans	43%
Pneumococcus	39%
Hemolytic streptococcus	25%

Baird[19] raises the question as to whether the major allergen in house dust might be bacteria. Extensive studies show that one of the largest elements in household dust is bacteria, especially staphylococci and streptococci, and it is interesting to note that bacteria are practically the only allergens which increase in quantity after reaching the body.

The preceding theoretical and practical evidence as well as the success of autogenous vaccine therapy in selected cases of bronchial asthma by the author and others[3,19,23] led to this study of 110 patients with allergic hyperplastic sinusitis. There was reason to believe that the infectious element was the predominant factor in producing their symptoms, although two-thirds of the patients showed positive skin tests to non-bacterial allergens. Over one-half of the cases had bronchial asthma as well as allergic sinus disease. Sixty-seven had hyperplastic mucosal changes without evidence of pus formation in one or more paranasal sinus as evidenced by trans-illumination, X-ray or at operation, and 35 had gross pus formation in at least one sinus in addition to the hyperplastic changes. Ten patients had what appeared to be normal sinus mucosa, but virulent organisms were cultured from all of them.

The respiratory mucosa of the upper and lower tracts have much in common and it is logical to assume that if asthmatic attacks could be controlled by reducing allergic responses in the pulmonary tract with vaccine therapy, then many of the allergic reactions in the sinus mucosa might be reduced to a minimum if the proper organism could be obtained for antigenic activity. If these allergic edema reactions could be controlled or minimized, the fundamental pathology of most chronic sinusitis would be checked.

Cultures which were taken with due precaution to prevent contamination were obtained from the middle meatuses of the nose in 80 patients, from bronchoscopic aspiration in eight, from tonsil tissue at operation in eight, from sinus membranes at operation in seven, from maxillary sinus irrigation in four and from the nasopharynx in three.

TABLE 2.

INCIDENCE OF BACTERIA CULTURED FOR USE IN VACCINES.

Hemolytic staphylococcus aureus	42
Steptococcus viridans	34
Pneumococcus (Types 12, 31, 9, 4, 5, 3, 22, 23, 14)	29
Nonhemolytic staphylococcus aureus	29
Nonhemolytic streptococcus	27
Neisseria catarrhalis	19
Nonhemolytic staphylococcus albus	17
Hemolytic streptococcus	16
Hemolytic staphylococcus albus	13
Diphtheroids	3
K. pneumoniae	1
Proteus	1
Total	231

Castellani, in 1905, showed that when two organisms were injected into an animal simultaneously, the antibody response to each organism was equal to that produced by injection of the individual organisms. A good general principle to follow, however, in the preparation of mixed vaccines is that they should contain as few different bacteria as possible, and only those should be incorporated in full dosage which are known or reasonably suspected to be pathogenic. The ideal method of preparation of an autogenous vaccine is one which modifies the organism as little as possible — *i.e.*, just enough to rob it of its disease producing qualities without destroying its immunizing capacity.

TABLE 3.

VACCINES.

Pure culture		20
Hemolytic staphylococcus aureus	11	
Hemolytic staphylococcus albus	5	
Streptococcus viridans	2	
Pneumococcus	1	
Nonhemolytic staphylococcus aureus	1	
Two organisms in vaccine		57
Three organisms in vaccine		29
Four organisms in vaccine		4

Vaccines were prepared for this study by the following method: A sterile swab of the secretion from the selected site was inoculated into nutrient broth made from brain heart infusion, a blood agar plate, and a modified chocolate agar plate. The latter was incubated in an atmosphere of 10 per cent carbon dioxide. Colonies of pathogens were picked from the blood agar plates and smears were made of these, and the growth in the broth, for identification. They were then transferred to nutrient broth from the agar plates and were grown separately for four to five days.

The suspensions were filtered through sterile gauze, after transfer to a sterile flask. The filtered suspension was centrifuged and the supernatant fluid was poured off. Saline was added to each tube and a dilution of two billion per cubic centimeter was made using McFarland nepholometer tubes for comparison. The cultures were then mixed proportionately so that the final concentration of the mixture was two billion per cubic centimeter.

The mixed saline suspension was then poured into small vaccine bottles. A second bottle was prepared to a dilution of one hundred million per cubic centimeter. These were submerged to the neck in a water bath at 60° C. for one hour on two successive days. Tricresol was added to a final concentration of 0.25 per cent and 1 cc. from each bottle was transferred to brain heart infusion broth for sterility.

Intradermal tests were done routinely with 0.05 cc. of the weaker dilution.

The problem of skin testing with bacterial extracts is a difficult one. There are divergent views on this subject, and the percentage of positive results reported varies widely. Some authorities[24] state that many nonallergic persons and a high percentage of patients suspected of bacterial allergy give cutaneous reactions to a wide variety of bacterial antigens. Touart[25] made the following report on intracutaneous testing with autogenous vaccines:

a. Reactions are constant.

b. Early positive wheal reactions persist in spite of prolonged vaccine treatment.

c. The late positive reaction grows less or disappears in the course of vaccine treatment.

d. Attacks of asthma have followed the injections of vaccine which gave positive early or late reactions.

e. Old quiescent test or treatment sites have been lighted-up by subsequent injections of the same vaccine.

Of the 110 patients tested intracutaneously in this series, only 24 gave early positive wheal reactions and 16 showed delayed (24 to 48 hours) skin responses. The dosage was increased 0.05 cc. or 0.1 cc. at five day intervals, depending upon the reactivity of the subject. General and local reactions occurred in approximately half the patients treated. It was not unusual to produce an asthmatic attack by increasing the vaccine dose too rapidly. Typical allergic flare-up in the sinuses was witnessed on several occasions as a reaction to the injection of autogenous vaccines in very sensitive individuals. No serious reactions were encountered.

Benson[4] states that the reactions of intradermal tests in conjunction with the systemic effects of bacterial inoculation form the most reliable guide to vaccine therapy in cases of bacterial hypersensitivity of the respiratory tract. His requisites for success in vaccine therapy are the proper choice of foci for culturing, careful isolation of the predominant organ-

ism as determined by plate counts and standardization of dosage by cutaneous tests and systemic reaction. Skin responses to streptococcus hemolyticus when present in his patients were usually comparable with the ordinary 24 hours' response to the Dick test and streptococcus viridans and the nonhemolytic streptococci usually elicited cutaneous responses which are similar to those obtained with streptococcus hemolyticus. Kraft, Mothersill and Nestman[21] showed that about 30 per cent of their subjects gave immediate urticarial reactions to intradermal injections of bacterial antigens.

Schonwald and Deppe[22] obtained positive results in 84.4 per cent of 155 cases treated with vaccines made from airborne bacteria which are nonpathogenic. They have demonstrated, by skin tests and positive results of treatment, that an allergy to these bacteria exists in many cases. There were 60 cases of allergic rhinitis reported in this group.

A small group of patients in the present report had been treated previously with mixed stock vaccines without much relief of symptoms. They universally improved with autogenous vaccine therapy. The determination of which type of vaccine to use depends upon clinical experience rather than laboratory observations. Since antigenic qualities do not remain unchanged by artificial cultivation as evidenced by loss of capsules, diminution of virulence, etc., it is highly advisable that the organisms causing the infection be obtained and included in the vaccine as quickly as possible. The existence of serologically specific types of bacteria is well known, as in the case of staphylococcus aureus, which was by far the commonest organism encountered in the respiratory tract in this series and others,[2] hence the injection of one strain in a vaccine may not protect against another. The use of a properly prepared autogenous vaccine reduces to a minimum the risk of using a different strain and makes it preferable to a stock vaccine for therapeutic purposes. The ability to utilize freshly isolated strains of virulent bacteria unchanged by prolonged cultivation is the main advantage of an autogenous vaccine in addition to the fact that freshly prepared vaccines are probably more antigenic than old stock vaccines.[26]

In this study, 90 cases of hyperplastic sinusitis and asthma were either improved or markedly improved with this type of therapy. The treatment of hyperplastic sinusitis with autogenous vaccines, either obtained from cultures of the nose, sinus membranes at operation, sinus washings, bronchoscopic aspiration, nasopharynx or tonsils, has proved to be of great value, particularly in those patients whose atopic allergies were controlled but whose symptoms continued or those who lacked skin sensitivity to nonbacterial allergens. Goodale[27] believed that an allergic individual who shows negative skin tests is sensitive to bacteria. The findings in this study corroborate this view.

SUMMARY.

A study is presented of 110 patients with respiratory allergy who had bacterial infection as the predominant cause of their symptoms. They were from six and one-half months to 84 years of age and had symptoms from one month to 50 years.

They were all treated with autogenous vaccines prepared from infected foci; the predominant organism being the staphylococcus. Mixed vaccines of two or more bacteria were used in the majority of patients, and the average period of treatment was one year. Local and general reactions were not uncommon. Intradermal skin tests were positive in 40 cases.

Improvement or marked improvement was observed in 90 patients.

The data included in this study is additional evidence that bacterial sensitivity plays an important rôle in the pathogenesis of chronic rhinitis, sinusitis and asthmatic bronchitis.

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INNER EAR DEAFNESS OF SUDDEN ONSET.*†‡

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This report concerns those cases of inner ear deafness of sudden onset which cannot be attributed to such well known causes as suppurative labyrinthitis, tumors, inner ear trauma or known toxic involvement. These cases are also clinically differentiated from Ménière's disease or idiopathic hydrops of the labyrinth by the fact that they are characterized by one attack only, with permanent impairment of function, whereas in Ménière's disease there are recurring attacks of vertigo and a deafness and tinnitus which fluctuate in severity from time to time.

Factual information as to the pathological process underlying the loss of function in these cases of sudden onset has been lacking since the incidence is low and the condition is not fatal. Our impressions as to the probable pathology have been gained from the postmortem examination of cases in the

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‡Since this report was written a fourth case of bilateral profound deafness and loss of vestibular function, associated with nonluetic keratitis, has been observed. A brief history follows:

W. S., a 31-year-old male, was in good health until 1939 when he developed blurring of vision. One month later he became dizzy and began to vomit. Within a few hours he noted deafness on the right. A diagnosis of keratitis and VIIIth nerve deafness was made.

In October, 1941, he again developed an alternating keratitis, loss of balance and sudden deafness on the left. A tentative diagnosis of C.N.S. lues was made despite negative blood and spinal fluid serology, and fever therapy was instituted without improvement. Since that time he has had a constant roaring in his head.

In October, 1948, he had a recurrence of the keratitis which responded again to atropine therapy but the hearing loss has been permanent. He was first seen in our clinic on Feb. 4, 1950, at which time his ear, nose and throat examination was negative. Apart from profound bilateral nerve type deafness and absence of caloric responses to ice water, this appears to be a fourth case of Cogan's syndrome which differs from the other two severe cases only in that this patient has had head noises.

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various stages of labyrinthitis, and from such conditions as leucemia and Paget's disease of the temporal bone which have undergone an acute inner ear disturbance in the terminal stages. Some evidence as to the susceptibility of the sense organ to chemical or toxic factors has also been gained from animal experiments.[1]

An acute inner ear disturbance resulting in permanent damage to function is known to occur in the course of some diseases, chiefly those of an acute infectious nature, such as mumps, typhus, Ramsey-Hunt syndrome, etc., but those cases in which no infectious process could be demonstrated have usually been considered as due to vascular disturbance in the labyrinth.

Some idea of the incidence of sudden deafness and vertigo in which the pathologic process is doubtful is indicated by the fact that the cases to be presented were selected in approximately a three-year period, whereas the number of cases of definite Ménière's disease during the same period is estimated at three or more times that number.

For purposes of comparison and discussion the report includes four cases (Cases 1, 2, 3, and 4) as examples of more common and well known types in which the acute inner ear disturbance was associated with some known infectious process and, therefore, less rare. The 12 succeeding cases seem to fall into one group in which the etiology is uncertain.

The last three cases seem to fit into a specific syndrome in which the inner ear disturbance was associated with an ocular disease.

Case 1: W. W., 58-year-old industrial engineer, had a history of bilateral otitis media with suppuration for one week in 1914. His hearing was subjectively normal. Dec. 31, 1946, he developed malaise and a mild temperature elevation. After two days he developed an earache on the right, which was followed in 24 hours by a serous drainage. A few hours after the drainage began he developed a roaring tinnitus and vertigo with nausea and vomiting. The nausea and vomiting subsided in three to four days and the vertigo became progressively less severe but was still aggravated by quick movements when seen four months later. At no time did he have any headache.

Ear, nose and throat examination four months after the onset of disease was normal apart from bilateral exostoses of the external canals.

Audiogram showed profound nerve type deafness on the right (see Fig. 1).

Spontaneous nystagmus could be demonstrated after head shaking, but no postural vertigo could be elicited.

Caloric response to ice water was absent on the right and was hyperactive on the left.

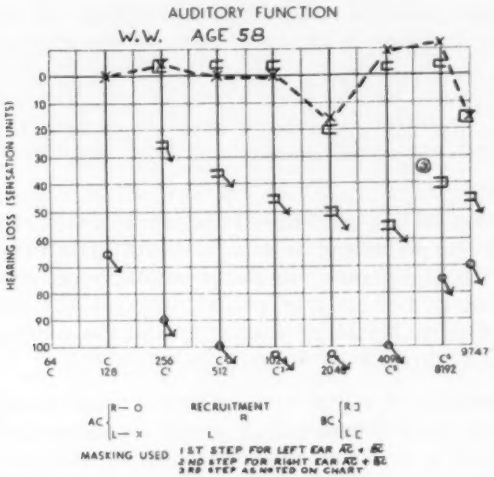

Fig. 1. Case 1. Acute suppurative otitis media followed in a few hours by tinnitus and profound deafness on the right with vertigo. The caloric response to ice water was absent on the right.

Comment: Although the history of frank suppuration from the ear was questionable, the diagnosis of diffuse labyrinthitis, serous or suppurative, seems definite.

Case 2: R. R., male, aged 27, was first seen on Jan. 26, 1939. At that time he gave a history of having had an acute osteomyelitis two years previously with marked temperature elevation, delirium, vertigo and hearing loss on the right. The ear did not discharge at this time. The vertigo subsided rapidly, but the deafness has been constant.

Examination of the nose and throat was normal. Both tympanic membranes were scarred but intact. An audiogram at the present time shows profound inner ear deafness on the right (see Fig. 2). The cold caloric response is normal in both ears and rotation tests are normal.

Impression: Metastatic embolus to the right inner ear with profound deafness and vestibular irritation.

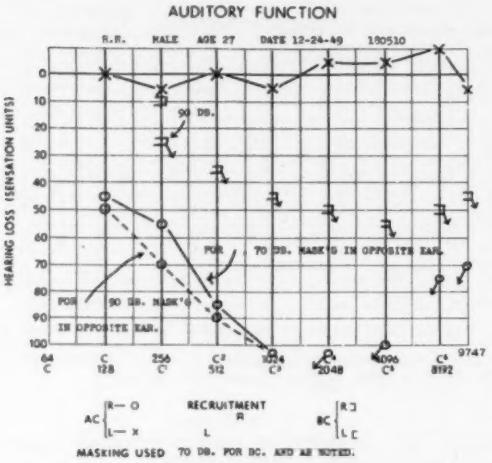

Fig. 2. Case 2. Sudden onset of vertigo and profound deafness on the right during acute osteomyelitis. Cold caloric responses were normal.

Comment: This case represents a sudden onset of deafness and vertigo without evidence of middle ear disease which occurred during an acute stage of osteomyelitis. The episode occurred before sulfonamide therapy was available. The preservation of normal vestibular function in the presence of profound deafness is a picture which is more suggestive of a toxic disturbance or of a metastatic embolus affecting the cochlear nerve than of a frank labyrinthitis.

Case 3: B. F., a 36-year-old housewife, gave a history of having had an eye infection and hearing loss, more severe on the right, 18 years before her visit on Dec. 18, 1946. Three months prior to this first visit, she had delivered a normal child, followed after one month by a sudden onset of head noises and almost complete bilateral deafness without vertigo. She had had bilateral chronic suppurative otitis media as a child.

Examination of the nose and throat were essentially normal. The tympanic membranes were intact but scarred. Audiogram revealed a marked bilateral nerve type deafness. Cold caloric responses were reduced bilaterally. The Kahn and Wassermann tests fluctuated between questionably positive and questionably negative. Antiluetic therapy was begun, but her hearing diminished still further while under treatment (see Fig. 3).

Impression: Late congenital lues. Marked bilateral nerve type deafness.

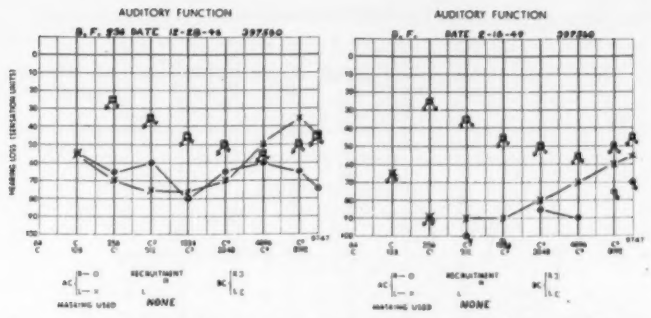

Fig. 3. Case 3. Sudden onset of severe deafness without vertigo one month after delivery of a normal child in a patient with congenital lues. The cold caloric responses were reduced bilaterally. The chart shows the progressive hearing loss during antiluetic treatment.

Comment: The history of sudden marked deafness three months before her first visit probably indicated a sudden exacerbation of deafness in her better hearing ear. The absence of vertigo at the time suggests a relatively mild exacerbation of the luetic process probably exerting a toxic effect on the auditory neural apparatus.

Case 4: E. L., a 20-year-old female student, has been under observation since childhood. She experienced an onset of sudden unilateral deafness while ill with epidemic parotitis when a child. There was no vestibular disturbance at any time.

Examination of the nose and throat was normal and both M.T.'s were normal in appearance. Auditory function was shown to be a profound deafness on the left, while the right was entirely normal (see Fig. 4). The cold caloric response was normal in both ears. No postural vertigo could be demonstrated.

Impression: Profound inner ear deafness, left, due to a toxic neuritis.

Comment: This case is included to illustrate the type of otitic complication which is frequently associated with mumps; unilateral profound and permanent deafness with little or no vertigo and no loss of vestibular function. This complication has occurred bilaterally in rare instances.

A toxic neuritis affecting the cochlear neural apparatus but sparing the vestibular sense organs is the usual interpretation but histopathological examinations have not been reported.

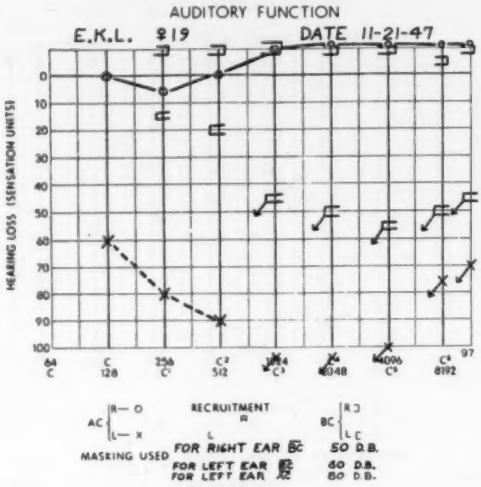

Fig. 4. Case 4. Audiogram of a patient who experienced sudden profound deafness without vertigo following mumps.

Case 5: A. N., a 28-year-old female, on Oct. 29, 1947, developed tinnitus and after a few hours deafness on the left. Twenty-four hours later she developed vertigo with nausea and vomiting. She was in good health at the time of onset but had had a severe cold one month previously. The patient never had had mumps but stated that at the time of her cold, her mother was recovering from a glandular swelling that was thought by her local physician to have been mumps. The patient was not seen until April 27, 1948. At this time she still complained of the deafness and a "sea-shell' tinnitus.

Examination of the nose and throat was essentially normal and the M.T.s were normal. Audiogram revealed the hearing to be normal on the right but profound nerve type deafness on the left (see Fig. 5). Cold caloric response was normal on the right and was slightly reduced on the left.

Impression: Profound inner ear deafness on the left of sudden onset, possibly due to subclinical mumps.

Comment: The ear complication in this case differs from that in Case 4 only in that vertigo was a prominent symptom at the time of the onset, and some loss of vestibular function has persisted.

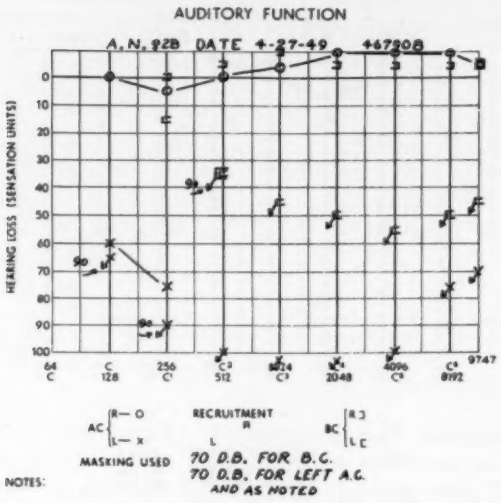

Fig. 5. Case 5. Sudden profound deafness and tinnitus on the left and vertigo without systemic disease one month after exposure to mumps.

The history of exposure to mumps one month previously suggests the possibility of a subclinical infection of this type with a complicating toxic neuritis of the VIIIth nerve.

Case 6: C. H., a 78-year-old female, was first seen March 5, 1949, with a history of bilateral decreased hearing for several years that was more pronounced on the right. History of previous suppuration in the right ear was given. Four weeks before her visit to the clinic she noted a low-pitched tinnitus on the left and a sudden marked hearing loss on that side.

Examination of the nose and throat was essentially normal. The right tympanic membrane was retracted and distorted, while the left was retracted. Hearing tests showed a moderate nerve type deafness on the right, and a profound nerve type deafness on the left (see Fig. 6). Cold caloric responses were normal on both sides. She gave a history of mild postural vertigo that was unchanged by the onset of sudden deafness on the left.

Impression: Moderate inner ear deafness on the right of long duration.

Profound inner ear deafness on the left of sudden onset. Etiology is doubtful.

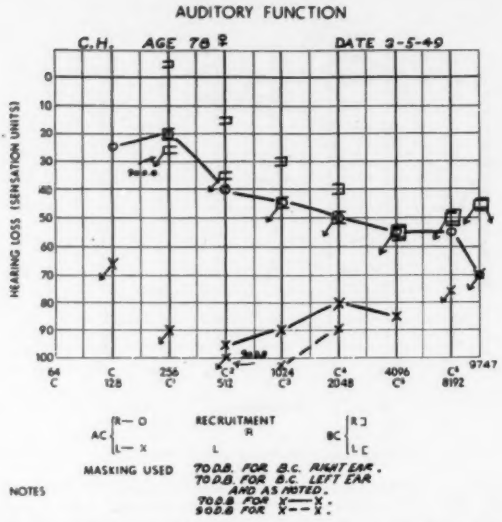

Fig. 6. Case 6. Sudden onset of tinnitus and profound deafness on the left without vertigo in a patient who had a previous bilateral deafness which was more severe on the right. The cold caloric responses were normal.

Comment: The sudden onset of profound unilateral deafness in the absence of any demonstrable infection at the age of 78 is suggestive of a vascular accident affecting the end-organ. Such an accident, in order to destroy hearing without interfering with vestibular function in any way, would necessarily be limited to the cochlear apparatus.

Case 7: M. Z. was a 54-year-old nurse. She was first seen April 15, 1938, stating that Jan. 15, 1938, she noted a click in her left ear which was followed by a persistent and severe hearing loss and a low-pitched tinnitus. She had no vertigo or diplacussis.

Nose and throat examination was essentially normal. Ear examination revealed normal M.T. bilaterally. Audiogram showed essentially normal function on the right but a profound inner ear deafness on the left (see Fig. 7). Cold caloric responses were normal.

Impression: Profound inner ear deafness, left, of sudden onset. Etiology is uncertain.

Comment: This case illustrates a sudden onset of unilateral profound deafness without vestibular disturbance. There was no history of any known infection at the time of onset.

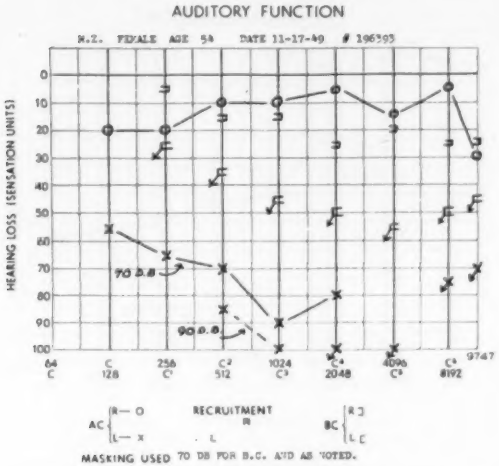

Fig. 7. Case 7. Sudden onset of severe deafness and tinnitus on left without vertigo. The cold caloric responses were normal.

The auditory complication was similar to that usually seen in mumps and is, therefore, suggestive of a toxic neuritis.

The age of the patient on the other hand might suggest a vascular accident. Such a vascular lesion would necessarily be localized to the cochlear apparatus.

Case 8: J. F. is a 36-year-old male who, on the night of June 23, 1949, while driving his car, had a feeling that something broke in his left ear and was followed by vertigo. He continued home and then vomited. He felt all right while lying down but vertigo returned on getting up. The following day he first noted deafness in the left ear. His vertigo had subsided in five days and at that time he was given injections of histamine and oral phenobarbital and nicotinic acid. Since his hearing did not improve, he presented himself at our clinic.

Complete neurological examination was normal apart from deafness on the left. Nose and throat examination was normal. The M.T. appeared normal. Audiogram revealed profound nerve type deafness on the left (see Fig. 8). Cold caloric response on the right was normal but there was no response to ice water on the left.

Impression: Profound deafness on the left with loss of vestibular function.

Comment: This case differs from the four previous cases in that the sudden onset of profound deafness was associated

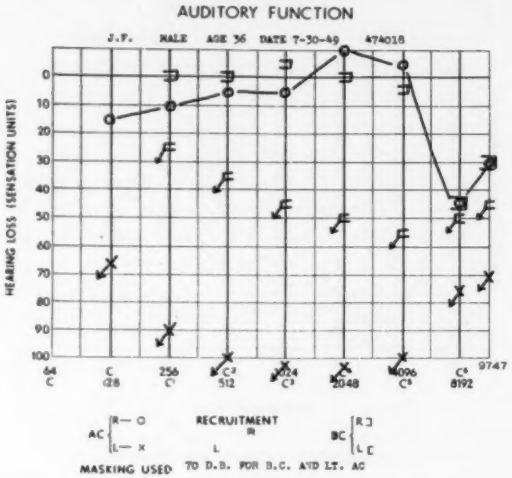

Fig. 8. Case 8. Sudden onset of profound deafness on the left and vertigo. The cold caloric response was absent to ice water on the left.

with a severe vestibular disturbance and permanent loss of vestibular function. There was no known infection at the time.

The diagnosis of a vascular accident in the labyrinth has been commonly made in such cases. The age of the patient, 36 years, is evidence against, but does not preclude, such a possibility.

Case 9: A. E. is a 49-year-old female who developed a sudden deafness with tinnitus in the right ear while sewing two weeks before her first visit on Aug. 28, 1949. That evening she developed vertigo with nausea and vomiting. The vertigo subsided in about three days. She had no known systemic infection at the time of the onset of symptoms. She also developed a blurring of vision at this time which persisted for two weeks. She had had intermittent drainage from the left ear since childhood.

Examination of the nose and throat was essentially normal. The right M.T. was normal, while the left was scarred but intact. Audiogram revealed a moderate nerve type deafness on the left and profound nerve type deafness on the right (see Fig. 9). Cold caloric response was normal on the left but was diminished on the right. There was no postural vertigo.

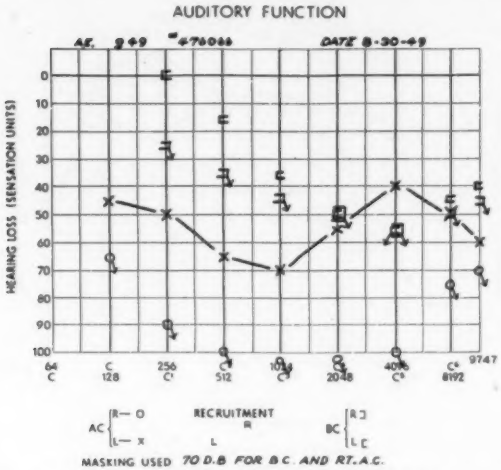

Fig. 9. Case 9. Sudden onset of profound deafness and tinnitus on the right and vertigo. The cold caloric response was diminished on the right. The left drum was scarred, due to previous suppuration.

Impression: Previous middle ear suppuration on the left with moderate nerve degeneration.

Profound deafness of sudden onset and moderate impairment of vestibular function on the right.

Comment: This case differs from Case 8 only in that vestibular function has been impaired rather than destroyed.

Case 10: M. F. was a 46-year-old female. She noted a sudden onset of deafness and roaring noises in the left ear in October, 1944, during a severe head cold. Noises began in the evening and the following morning she developed dizziness, nausea and vomiting which kept up for one week. The vertigo disappeared gradually but the deafness and tinnitus have persisted. There was no history of postural vertigo after the first attack had cleared, although a severe, sudden noise has caused momentary dizziness on two occasions since then. She had mumps at the age of 18, also measles, chicken pox and whooping cough.

Examination of the nose and throat was essentially negative and the M.T. appeared normal. Audiogram revealed normal hearing on the right and a moderate nerve type deafness on the left (see Fig. 10). Cold caloric responses were normal in both ears. No postural vertigo could be demonstrated.

Impression: Unilateral nerve type deafness of sudden onset with mild vestibular symptoms.

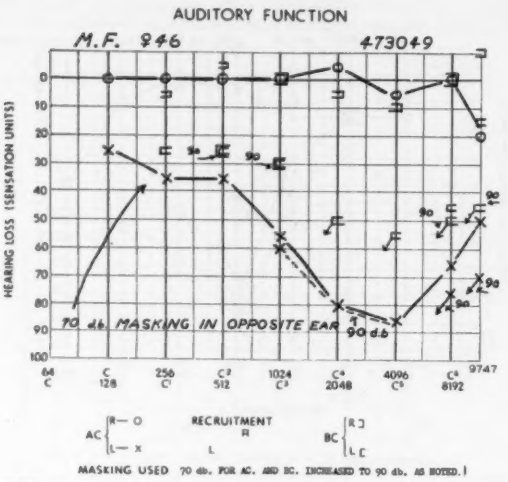

Fig. 10. Case 10. Sudden onset of vertigo and tinnitus and moderate deafness on the left during a severe head cold. The cold caloric responses were normal.

Comment: This case would appear to belong to the same group as Case 9 and to differ only in the matter of degree. Although a vascular accident in the inner ear is frequently assumed in such cases the fact that symptoms began during a severe cold raises the question of a toxic neuritis.

Case 11: S. N., a 29-year-old male, was first seen Dec. 2, 1949, and stated that 18 months before he had become dizzy while working in the sun. He was told that he had a "heat stroke" and was sent home. That evening he noted that he had a ringing in his left ear and that he could not hear the telephone with the left ear. The vertigo subsided quite rapidly, although he still describes what may be a mild postural vertigo. The hearing and tinnitus have not changed.

Examination of the nose and throat was essentially normal. The M.T. appeared normal to inspection. Audiogram showed the hearing to be normal on the right but a nerve type deafness on the left (see Fig. 11). Cold caloric responses were normal in both ears.

Impression: Nerve type deafness of rapid onset, left.

Etiology: Uncertain.

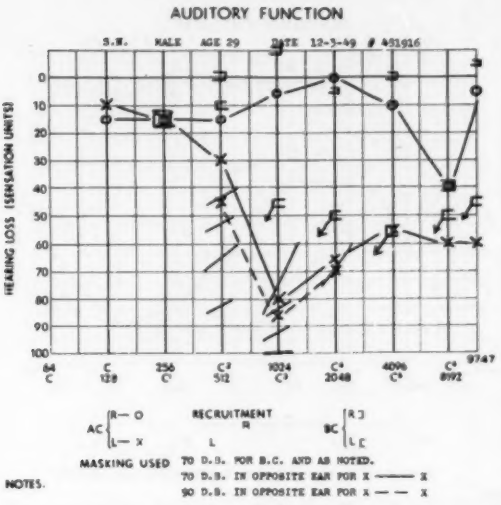

Fig. 11. Case 11. Sudden onset of vertigo followed by tinnitus and moderate deafness on the left while working in the sun. The patient still describes a postural vertigo but cannot be demonstrated. The cold caloric responses were normal.

Comment: This case apparently corresponds closely to Case 10 with the one exception, that the vertigo was complained of as a postural symptom for 18 months after the onset.

This patient was only 29 years of age and showed no evidence of systemic disease. A vascular accident such as a thrombosis or hemorrhage is, therefore, improbable.

Case 12: C. V. is a 27-year-old female who went to bed in the evening with a mild upper respiratory infection. On arising in the morning she had vertigo with nausea and vomiting. During the day when using the telephone she noted that she was unable to hear with the right ear. The vertigo passed away in about 24 hours, but for about one month she noted that she became dizzy very easily. She described no definite postural vertigo, but she believes that her hearing does fluctuate a little and that there had been some improvement.

On examination, Feb. 12, 1948, which was two weeks after the onset, the nose and throat examination was normal and the M.T. appeared to be normal. Audiogram revealed normal hearing on the left and a high

tone nerve type deafness on the right (see Fig. 12). Cold caloric responses were normal on the left and reduced on the right. There was no postural vertigo demonstrated.

Impression: High tone nerve type deafness of rapid onset of uncertain etiology.

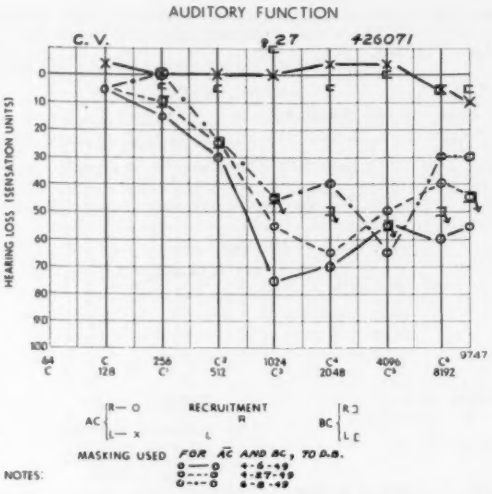

Fig. 12. Case 12. Vertigo and sudden moderate deafness on the right during a mild upper respiratory infection. The cold caloric response was reduced on the right. The chart shows the spontaneous hearing improvement in two months' time.

Comment: The onset of the unilateral deafness with vertigo was in this case associated with a mild upper respiratory infection. The severity of findings and the course of the disease corresponds closely, however, to other cases reported in this group and a similar etiology is, therefore, probable.

The audiogram illustrates the degree of recovery which occurred spontaneously.

Case 13 (previously reported in *Annals of Otology, Rhinology and Laryngology*, 56:3:541, Sept., 1947): L. S., female, aged 40, experienced a sudden onset on March 1, 1946, of screaming and whistling noises in the right ear, associated with deafness and a gradual onset of vertigo which reached a maximum about one week later. The vertigo has been postural, brought on especially by placing her head backwards, lying on one side, and after sitting up. It has not decreased in the period of one

year since the onset. She is unable to tolerate high-pitched loud sounds or music. She has had no headaches. Her ears were normal previous to this episode. There has been no appreciable change in the symptoms during the past year.

The general examination, including the neurological, was essentially negative.

The nose and throat examination was negative. The tympanic membranes were normal. There was moderate high-tone nerve deafness in the right ear (see Fig. 13).

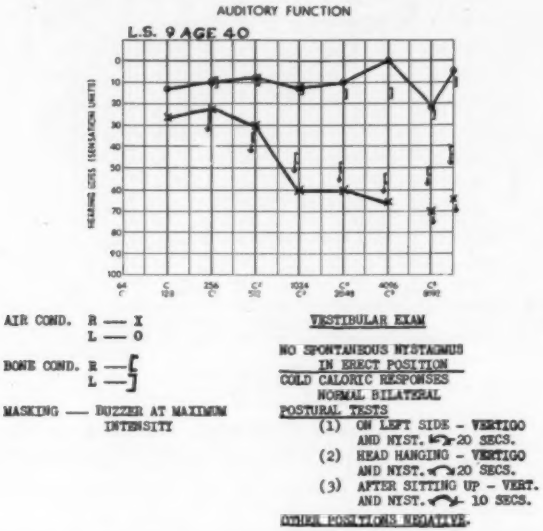

Fig. 13. Case 13. Sudden onset of tinnitus and impaired hearing in the right and gradual onset of vertigo. Postural vertigo was present one year after the onset.

No spontaneous nystagmus was present in the upright position. The fistula test was negative. There was a normal cold caloric response in each ear. The postural tests revealed a marked vertigo and nystagmus in the head hanging position, on the left side, and on sitting up. The positional nystagmus was counterclockwise in the left lateral position but clockwise in the head hanging position and after sitting up quickly.

Impression: High tone nerve type deafness, right, of uncertain etiology.

Case 14 (previously reported in the *Annals of Otology, Rhinology and Laryngology*, 56:3:541, Sept., 1947): H. R., female, aged 35, complained of deafness and noises in the left ear and dizziness. The symptoms came on suddenly three years earlier.

Vertigo was accompanied by vomiting at the onset. After about two weeks the dizziness had receded in severity and occurred only in certain positions. There were no further attacks, but the postural vertigo has persisted without further improvement until the present. It is noted particularly on lying on the left side. A buzzing noise in the affected ear and a sense of pressure or blockage varying in severity from time to time has been troublesome. Her hearing is greatly depressed.

There were no ear symptoms previous to the onset of the present complaints.

The general examination was essentially negative.

Local ear, nose and throat examination showed normal tympanic membranes. The right maxillary sinus was filled by a large polyp projecting into the nasal passage. There was no evidence of suppuration.

The auditory examination revealed profound nerve deafness on the left side but some residual hearing was present with adequate masking. There was normal hearing on the right (see Fig. 14).

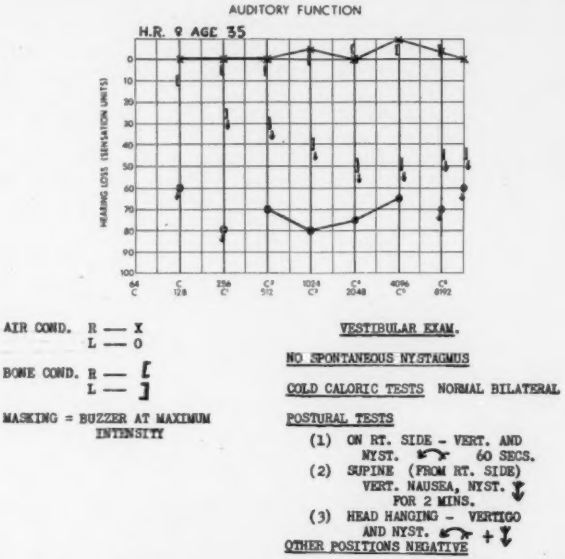

Fig. 14. Case 14. Sudden onset of tinnitus and severe deafness on the left and vertigo three years previous. Postural vertigo and unilateral tinnitus and deafness persist.

There was no spontaneous nystagmus in the upright position. The fistula test was negative. There was a normal cold caloric response in both ears. In the postural tests vertigo was accompanied by positional nystagmus in the supine and head hanging positions.

A radical antrum operation on the right revealed the antrum to be filled by polypoid and cystic tissue.

Nine months later the complaints referable to the left ear remained unaltered.

Impression: Profound deafness of sudden onset, left.

Etiology: Uncertain.

Comment: Cases 13 and 14 are very similar and can be discussed together. The possibility of a thrombosis due to an arteriosclerotic lesion affecting only the cochlear division of the arterial suply has been suggested to explain sudden deafness on the basis of vascular distribution to the labyrinth.

While a vascular accident might offer the best explanation for the apoplectic type of a Ménière's syndrome occurring as a single episode in a previously healthy ear after middle age, factual information regarding the pathology is at present inadequate. A toxic neuritis sometimes cannot be differentiated.

Case 15: H. M., female, aged 37, was first seen on Sept. 25, 1948, and at that time gave a history of severe right parietal headache one evening in May, 1945. She went to bed but was awakened by vertigo, nausea and vomiting. Three to four days later she developed a tinnitus and deafness in the right ear. She had no systemic disease at the time of onset. The vertigo subsided in a few days except when she assumed certain positions. This postural vertigo persisted for about three years and has gradually disappeared. After one year she believed the hearing began to return; but the tinnitus persisted, and loud sounds continued to cause an unpleasant sensation in the right ear. Her past history is contributory inasmuch as she was always susceptible to motion sickness and that she had migraine headaches since 1942, which seemed to have been controlled by histamine.

Examination of the nose and throat was normal, and the M.T. appeared normal. Audiogram revealed a high tone nerve deafness on the right (see Fig. 15), while hearing was normal on the left. The cold caloric response was normal on the left but was reduced on the right. No postural vertigo could be demonstrated on examination.

Impression: High tone nerve deafness and reduced vestibular function on the right of uncertain etiology.

Comment: This case demonstrates a vestibular disturbance associated with headache and unilateral deafness of rapid onset. The hearing loss and vestibular dysfunction were permanent. Postural vertigo persisted for about three years. The case corresponds closely to Cases 13 and 14, except for slight difference of degree.

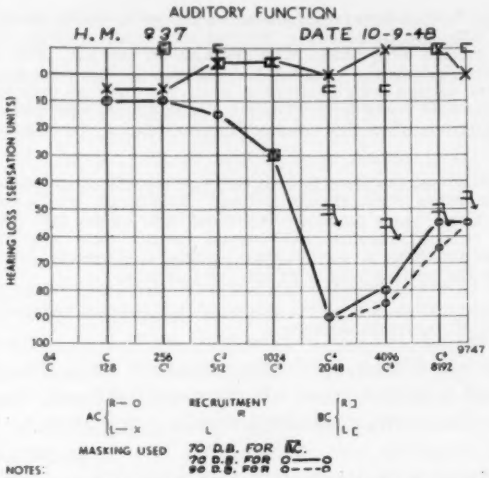

Fig. 15. Case 15. Sudden onset of tinnitus and deafness on the right, along with vertigo four years earlier. Postural vertigo persisted for three years, decreasing in the fourth year. The cold caloric response was moderately decreased on the right.

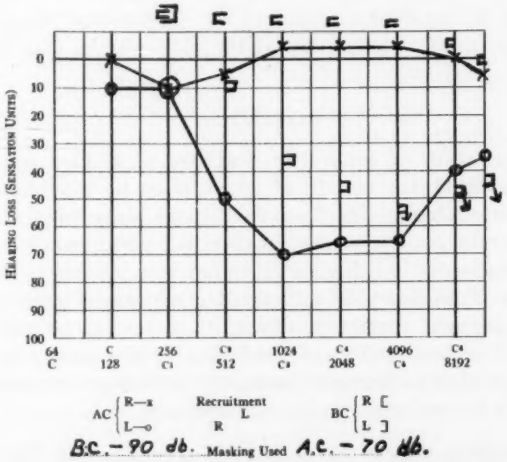

Fig. 16. Case 16. Rapid onset of tinnitus and moderate deafness on the left with vertigo. A postural vertigo persisted over one and one-half years. The cold caloric response was slightly reduced on the left.

Case 16: V. D., 24-year-old female, was in good health until July, 1946, at which time she developed a tinnitus that was followed in about two hours by vertigo with nausea and deafness on the left. The vertigo persisted about 10 days and required bed rest for the first two days, followed by postural vertigo which persisted until the time of examination over one and one-half years later. Positional nystagmus was demonstrated at the examination. Some spontaneous improvement in hearing was observed.

Examination of the nose and throat was essentially normal. The tympanic membranes were grossly normal. Audiogram was normal on the right, but there was a moderate nerve deafness on the left (see Fig. 16). The cold caloric response was normal on the right but was reduced slightly on the left. A postural nystagmus was present for 15 seconds only when the patient was lying on her left side.

Impression: High tone nerve deafness and reduced vestibular function on the left of uncertain etiology.

Comment: This case closely resembles Cases 13, 14 and 15 which had a sudden onset of inner ear deafness, tinnitus and vertigo. The vertigo subsided within a few days to become a postural symptom only, which was known to persist for as long as three years; however, this patient was only 24 years of age and was in good general health. A vascular accident in the inner ear could scarcely be considered as a likely etiologic factor. Although there was no history of any known infection at the time of onset of the episode the possibility of a toxic process affecting the inner ear must be considered.

DISCUSSION.

Sixteen cases of inner ear deafness of sudden onset have been presented, the last 12 of which must be considered as of indefinite etiology. The first four cases were presented only to illustrate types of sudden inner ear disturbance in which the etiologic relationship to infection was evident. These are all cases of permanent unilateral deafness of sudden onset with or without vestibular disturbance, occurring as a single attack. The localization of the lesion to the inner ear seems definite in view of the association of vestibular symptoms with moderate to profound unilateral deafness.

The sudden onset, the single attack only, and the permanent loss of function are sufficient to differentiate these from Ménière's disease (idiopathic hydrops of the labyrinth). In

Ménière's disease there are remissions and exacerbations of tinnitus and low-tone deafness, and usually progression of the deafness over a period of years. Although there are repeated attacks of vertigo, vestibular function as shown by the cold caloric tests is usually normal in the early stages. Because of these facts it seems improbable that the same pathologic disturbances occur in both diseases or that they are due to the same etiologic factor.

Deafness of sudden onset, sometimes associated with vertigo, is known clinically to occur on a toxic basis as a complication of some infectious diseases. These are mainly virus in type. Involvement of the vestibular apparatus has not usually been proportionate to the auditory disturbance and may even be absent. There is, however, no constant pattern as regards proportion of hearing loss to vestibular dysfunction as a result of a toxic process.

Vascular disease of various types is also considered a cause for deafness of sudden onset with or without vestibular disturbance.

Hemorrhage is perhaps considered first when vascular disease is thought to be the etiologic factor. Several of the patients reported here were still in their twenties and in apparent good health; therefore, hemorrhage is an improbable explanation. None was known to have had blood dyscrasias, and none had been exposed to head trauma.

Patients who had developed vestibuloauditory symptoms in association with leucemia[2] and whose temporal bones had been subsequently examined were found in some instances to have a serofibrinous exudate in the labyrinth and in some to have fibrosis and a labyrinthitis ossificans. These findings in cases of leucemia appear to have been the main basis for the diagnosis of hemorrhage in the labyrinth in cases such as those presented in this report.

Thrombosis, occlusion or spasm with resulting ischemia are other vascular conditions that are not easy to differentiate. The vascular supply of the cochlea is such that a thrombus

might affect the entire auditory sense organ and theoretically might account for deafness without vertigo. The majority of cases presented here, however, have shown varying degrees of involvement of both auditory and vestibular function and, therefore, are not explainable on the basis of vascular occlusion; also, the occurrence of the disease before 30 years of age in a significant proportion of cases is evidence against vascular accident.

A vasomotor disturbance as postulated by Brunner[3] must also be considered. The pathologic findings in a case of Paget's disease of the temporal bone which had experienced a severe vestibuloauditory disturbance shortly before death were described by him as otitis interna vasomotoria. While there seems to be little evidence to indicate that the type of pathologic change demonstrated in advanced Paget's disease might occur in otherwise normal young adults, it must be admitted that a vasomotor disturbance offers a possible explanation.

In searching for the etiologic factor of Ménière's disease, allergy has been suggested and has received much consideration. Rather infrequently has there been direct evidence to associate atopic allergy with Ménière's disease. Intrinsic or physical allergy has been postulated as an etiologic explanation. Inasmuch as these cases are characterized by a single episode in a lifetime, an allergic factor appears unlikely.

THE SYNDROME OF DEAFNESS AND VERTIGO AND NONLUETIC INTERSTITIAL KERATITIS AND IRITIS.

Case 17: H. N., 38-year-old female, noted reddening of her eyes in November, 1943, which subsided over a period of three months. She had a slight but daily temperature elevation for one month beginning in December, 1943. About the time that her eyes improved she began to have tinnitus on the left, which soon became bilateral and was associated with bilateral deafness and vertigo on movement.

She was first seen March 14, 1944, at which time the general physical examination was essentially normal. There were no abnormal findings in the nose and throat and the M.T. appeared normal. Neurological examination was also normal apart from the vestibuloauditory findings. Audiogram revealed a bilateral nerve type deafness (see Fig. 17). Cold caloric responses were hypoactive in both ears. The eye disease was

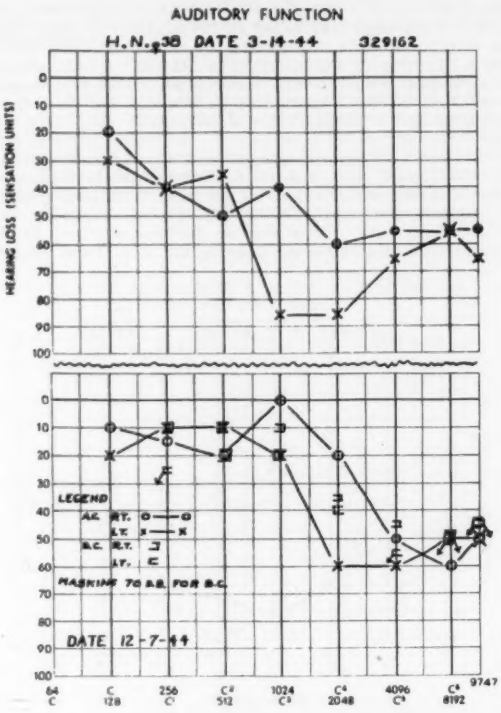

Fig. 17. Case 17. Bilateral nerve type deafness and tinnitus of rapid onset with vertigo and reduced cold caloric responses associated with bilateral iritis. The spontaneous recovery is illustrated.

diagnosed as bilateral iritis with secondary glaucoma on the left. She had been followed since that time and there has been some recovery of hearing.

Impression: 1. Bilateral nerve type deafness with reduced caloric responses. 2. Bilateral iritis with secondary glaucoma on the left.

Case 18: J. M., male, aged 27, was well until the morning of Oct. 20, 1948, when on arising he noted nausea, vomiting and vertigo. Twenty-four hours later he noted bilateral tinnitus and deafness which was progressive for 24 hours. The vertigo subsided in three days and by the end of one week the hearing was normal on the right and but slightly impaired on the left. He was well until Feb. 1, 1949, when he again noted vertigo with nausea and vomiting for a few hours. A lumbar puncture done at that time was reported to be normal. Five days later

he developed pain and "conjunctivitis" in the left eye which lasted three days, but one week later was noted in the right eye. After that time the complaint alternated from one eye to the other. Feb. 25, 1949, he began to develop a blurring of vision between attacks of pain and a profound deafness developed. Skull X-rays, EEG, blood, urine and complete neurological examination were reported to be normal. He was then placed on daily histamine and vitamins without change.

He was first seen in our clinic April 6, 1949, at which time the general physical, neurological and nose and throat examination were normal. The tympanic membranes appeared normal. The audiogram showed a profound bilateral deafness (see Fig. 18). Caloric response to ice water was absent in both ears. There was no postural vertigo. Examination of the eye was reported as showing a nonluetic interstitial keratitis.

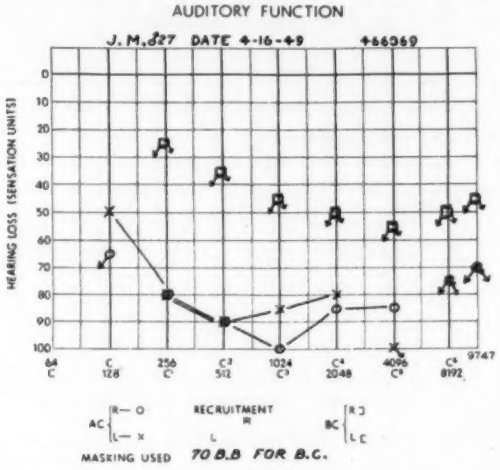

Fig. 18. Case 18. Rapid onset of bilateral deafness and vertigo associated with a bilateral nonluetic interstitial keratitis. The cold caloric responses to ice water were absent.

Impression: Nonluetic interstitial keratitis associated with profound bilateral inner ear deafness and loss of vestibular function.

Case 19: F. Di P., male, 26 years of age, developed in September, 1946, a sudden "conjunctivitis" with a right subconjunctival hemorrhage. The eye pain cleared in two days apart from a blurring of vision but was followed by bilateral tinnitus and 12 hours later by vertigo with nausea and vomiting. Three days later there was a rapid onset of profound deafness on the right and a severe loss of hearing on the left. He was treated with sulfonamides and penicillin for three and one-half weeks, followed by streptomycin (period unknown), after which the residual hearing on the left was lost. In March, 1947, he developed cardiac symptoms, palpi-

tation and chest pain. The condition was controlled, but in November, 1948, he developed hemoptyses which subsided when the cardiac condition was again controlled.

When first seen in our clinic, Feb. 10, 1949, his general physical examination revealed an enlarged but compensated heart. Neurological examination was essentially negative. Eye consultants diagnosed a bilateral nonluetic interstitial keratitis. The nose and throat examination was essentially normal and the M.T. were normal. Audiogram showed a profound bilateral inner ear deafness (see Fig. 19). Cold caloric response was absent to ice water in both ears. There was no postural vertigo.

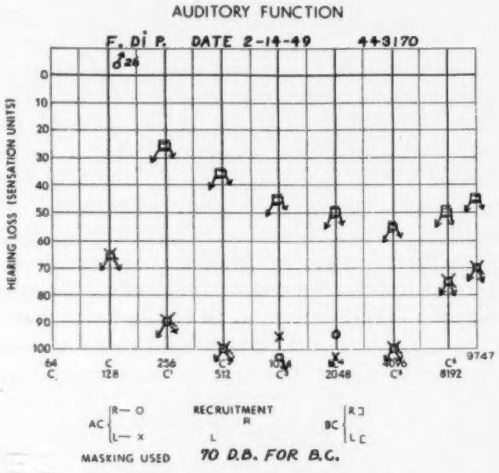

Fig. 19. Case 19. Rapid onset of bilateral deafness and vertigo associated with a bilateral nonluetic interstitial keratitis. The cold caloric responses to ice water were absent.

Impression: Bilateral nonluetic interstitial keratitis with profound bilateral inner ear deafness and loss of vestibular function.

Comment: Cases 17, 18 and 19 are all very similar and apparently belong to a specific syndrome. They differ from the previous cases in that the inner ear dysfunction is bilateral and associated with eye complications. Two of the cases had a single attack while the third had two attacks. Only one case had partial recovery of both ocular and inner ear function.

This syndrome differs from the syndrome of uveoparotid fever, which is usually characterized by a chronic painless parotitis, uveitis, facial paralysis and other neurologic changes in about 50 per cent of the cases.[4] The neurologic changes are mostly peripheral in type and are usually unilateral. Dysphagia is common and a nerve deafness is occasionally seen. The syndrome is frequently seen in patients with Boeck's sarcoid and the presence of lesions, on biopsy of the parotid, which resemble noncaseating tuberculosis has led many to believe that uveoparotid fever is a complication of Boeck's sarcoid.[5] The cases reported here show only an interstitial keratitis or iritis and inner ear dysfunction of sudden onset without neurologic manifestations. These findings do not correspond to those of uveoparotid fever and suggest a different syndrome. Cogan reported a series of four cases and cited a fifth which had been previously reported in which there was a nonluetic interstitial keratitis and inner ear dysfunction.[6] The order of appearance of symptoms was variable and the duration of progression was up to several weeks. Only one of the five cases demonstrated improvement and only after a period of years. In our cases as well as those of Cogan the diagnosis of lues appeared to be excluded. One case received aureomycin therapy without improvement, but this was over two months after the onset. It is apparent that our three cases as well as those of Cogan belong to the same syndrome. Although the etiology is uncertain, the inflammatory nature of the disease seems definite. Some type of virus disease seems most probable.

SUMMARY.

Sixteen cases have been presented of unilateral inner ear deafness of sudden onset with or without vestibular symptoms. The first four cases represent types in which the ear complication was associated with a systemic disease. Case 1 was apparently a diffuse labyrinthitis secondary to middle ear infection. Case 2 was probably a metastatic involvement of the inner ear during acute osteomyelitis. Case 3 was toxic in nature due to an exacerbation of a congenital luetic process in the ear. Case 4 represents the toxic neuritis of the audi-

tory neural apparatus associated with epidemic parotitis. The next 12 cases of unilateral sudden inner ear deafness with varying degrees of vestibular disturbance are of uncertain etiology. It cannot be definitely assumed that all 12 cases have the same etiology, but the similarity of this clinical picture strongly suggests that they are of the same type. A vascular accident in the labyrinth has commonly been the explanation in cases such as these. The high incidence in normal adults below 30 years of age argues against such a vascular accident. A vasomotor disturbance affecting the inner ear has been suggested and although hypothetical must be given consideration. Toxic involvement of the inner ear is a well known occurrence in infections of various types, some of which have been illustrated. In these cases of toxic neuritis, no common pattern exists as to proportionate vestibular and auditory dysfunction.

A toxic neuritis would provide a favorable explanation for the 12 cases presented of indefinite etiology and must be seriously entertained. The onset following an acute upper respiratory infection in two cases may give some support to this explanation.

The last three cases demonstrate a specific syndrome of bilateral inner ear dysfunction associated with an inflammatory ocular disturbance. Two of these had profound loss of auditory and vestibular function while the third had a less severe disturbance followed by partial recovery of function. The inner ear disturbance in this syndrome can be assumed to be toxic in nature, associated with an inflammatory process of unknown etiology.

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SURGICAL CORRECTION OF ANTERIOR WEB OF THE LARYNX.*†

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Anterior web of the larynx has always been a difficult problem. Until about 1880, when Schroetter,[20] of Vienna, started using indwelling cores of hard rubber and tin, the surgical treatment of stenosis of the larynx was considered hopeless. As recently as 1942, Loch and McHugh[13] state that surgery of anterior webs should never be attempted for improvement of voice only. They surveyed the literature to that time and were apparently impressed by the high degree of failure with any of the variations of the pioneer methods. These were three: repeated bouginage, splitting of the larynx by external operation with excision of scar, usually with the placement of a skin graft held by a "stent" of some sort, or the prolonged wearing of an O'Dwyer[16] tube or some modification of it. The methods were often combined.

Delsaux,[6] Schmiegelow,[19] Negus[15] and later Arbuckle,[1] Lynch,[12] New,[8] Figi[9] and others write of external methods, certainly inevitable in those cases of extensive trauma or obliteration of the lumen. Negus gives a good review of the problem to 1938. Hard and soft rubber tubes and plugs, metals including tantalum, and most recently acrylic forms have been employed, with and without skin grafts. For congenital or other webs of the thinner variety, Jackson, Clerf,[5] Tucker[21] and others advocate repeated bouginage sometimes after incision along one cord edge or in the midline with special knives

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or radio knife. Triangular bougies and heated bougies have been used. Jackson urges conservatism in children, for use of the larynx stimulates its growth.

Haslinger,[10] using his directoscope, placed a silver plate between the severed cords and anchored it there with a wire

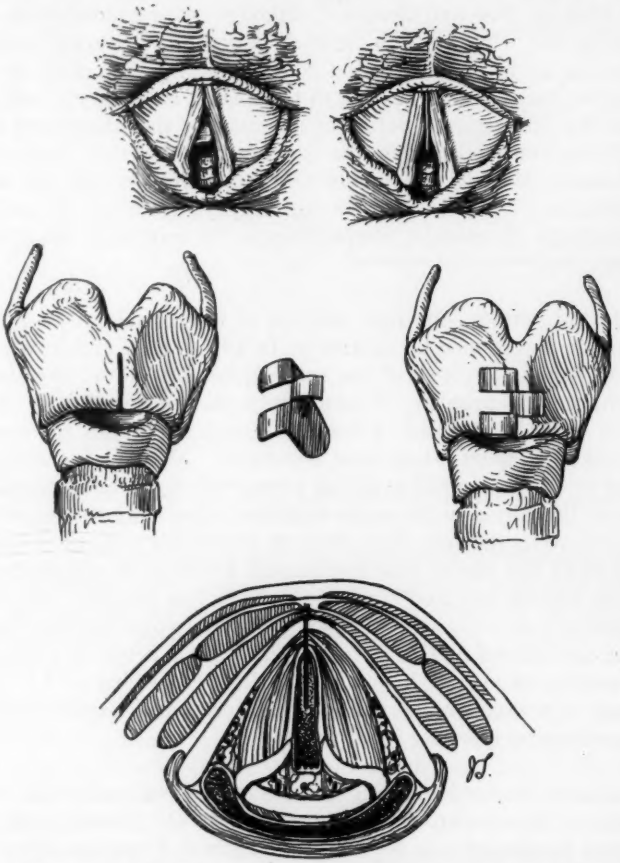

Fig. 1. Lower figure is a horizontal section at the level of vocal processes of arytenoids. It is schematic, since both right and left tabs would not show in one section. Note that the thyrohyoid muscles are sewed together in the midline to prevent outward displacement of the plate.

through the thyroid cartilage at the anterior commissure fastened to a button on the skin. In one case the plate cut through the thyroid cartilage and had to be removed by external incision. In another of the patients, carcinoma of the larynx was diagnosed six years later. Chondritis or the fear of it limited the use of this promising method. It was revived in 1948 by Poe and Seager,[17] although their modification of having the wire hug the inferior edge of the thyroid rather than to go through it would seem to permit the plate to be pushed backward by healing in the angle. Haslinger's method had the fundamental defect of all schemes that disregard the relative motion between the larynx and the skin and subcutaneous tissue. The earlier workers certainly did not lack ingenuity. We can profit by their experience with the added advantage of modern chemotherapy to minimize complications.

In my opinion, the real solution of the anterior web problem was envisioned by Iglauer.[11] In 1935, in an article which is an excellent review of the subject, he pointed out the similarity to the problem of syndactyly and reasoned that if a tract could be established first at the extreme angle, the cords would stay apart when later separated. All gaping incisions heal by fibrin strands bridging across the angle and contracting to literally sew the edges together, advancing the angle as this is accomplished. The place to check this undesired healing is at the apex. The mechanical problem of congenitally fused fingers defeated surgeons until it was realized that the important area was at the apex. When an epithelialized tract was established at this point by allowing a seton-like suture to remain in place a long time (as in the piercing of an earlobe), a subsequent incision to the free edge would remain open despite apposing raw surfaces.

Iglauer inserted the ring from a watch chain through the anterior commissure in one case. It was the type of ring in which a segment can be retracted against a spring. Due to technical difficulties the case was not entirely successful but the principle appealed to me as being the correct one; how-

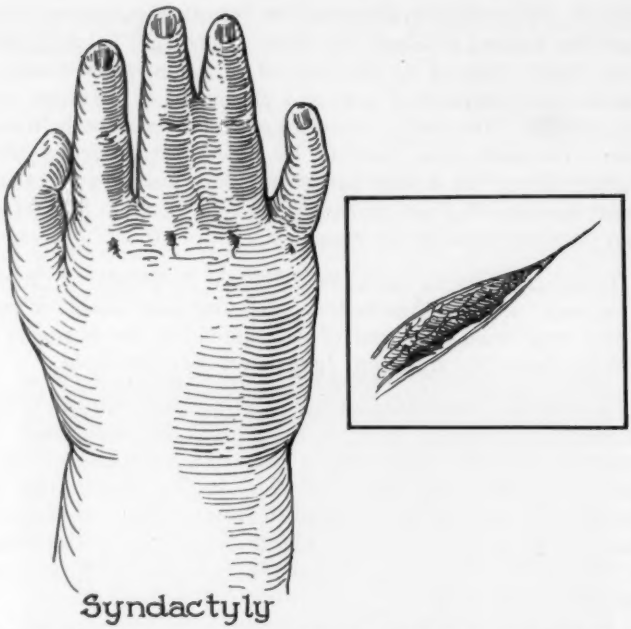

Fig. 2. Inset shows typical wound healing with fibrin strands bridging across the angle. Their contraction pulls the edges together, advancing the angle and literally sewing the wound closed.

ever, in the cases I have seen, no matter how deceptively thin the crescentic free edge of the web appeared, the webs were all too thick at the base for a small ring. A safety pin, closed subglottically, would be better, but I have not yet had a chance to try it.

In our experience congenital web of the larynx is not a problem commonly met. Stenosis from scar tissue web seems to have been more frequent when typhoid and lues were not so well controlled and when intubation of the larynx was freely employed. In the early days of my practice I inherited two adolescents who had undergone years of laryngeal dilatation by bouginage in an effort to get rid of tracheal cannulae. Both were the result of dense scarring from improper intu-

bation. I succeeded in one case but the other patient gave up. Another patient at about the same period had a luetic stricture, finally treated by the method of Schmiegelow; that is, fissure with excision of scar and placement of a rubber tube secured by a silver wire transfixing the tube and both thyroid alae. The tube was removed by direct laryngoscopy three months later; but a long period of dilatation with the triangular bougies of Clerf proved necessary, because the cricoid had been involved in the chondritis.

These experiences made me reluctant to employ laryngeal bouginage or other methods of fighting scar tissue if any better way could be found of accomplishing the purpose. It was at about this time that Iglauer's paper appeared.

The case to be described is not one of a thin web but rather of stenosis resulting from dense scar tissue occupying the anterior one-third of the larynx. It extended subglottically a full centimeter below the level of the cords, fusing them together. The method I employed developed from an effort to apply the principle described, that is, to interfere with healing at the apex. Others as noted above had attempted this, but not all have stressed the importance of the apex.

REPORT OF CASE.

Mrs. B. was first seen 18 years ago when in her late thirties. She was, and still is, a saleswoman in a department store. She complained of loss of voice of three months' duration. She had a sessile papillary growth exactly in the anterior commissure, the attachment extending subglottically. This was removed by indirect laryngoscopy by my associate in September, 1931, and in the succeeding weeks several additional bits of tissue were taken for biopsy. There were some irregular nodules on each cord and the anterior one-third of the left cord showed a pale subglottic swelling. Some sections showed giant cells and epitheliod cells which Dr. Ophuls considered suspicious of lupus or tuberculosis. Voice rest and quartz light treatment to the larynx were employed. Some months later the anterior commissure was filled with granulations forming a web. This was punched out in August, 1932, and February, 1933. The probability of tuberculosis seemed more and more remote as the patient's general health remained good. She had some old scars at the apices, but repeated sputum examinations were negative. The Wasserman was negative.

She had returned to work although the voice was very poor. Through the intervening years she has been seen from time to time for other things, the web gradually growing thicker and denser. Not until 1948 was the patient desirous of having anything further done to the larynx.

By this time she realized that she was getting very tired from the continual effort of talking, required in her work. Dyspnea was beginning to be evident. The anterior one-third to two-fifths of the larynx was filled in by dense scar tissue.

In December, 1948, the following operation was devised and carried out under local anesthesia: a transverse incision was made through skin and platysma in a skin fold at mid thyroid-cartilage level. The ribbon muscles were separated in the midline down to perichondrium, which was incised in the midline but not otherwise disturbed. A thin bladed circular saw about 2 cm. in diameter was used to make a midline slot in the lower two-thirds of the cartilage only. It was a saw intended for the "Handee" tool but the mandrel fits the usual dental handpiece. The slot was made from the cricothyroid membrane upwards but stopped short of the thyroid notch, so as to leave a bar of cartilage connecting the two alae. The purpose was to prevent any overriding or displacement which might have occurred if the separation had been complete.

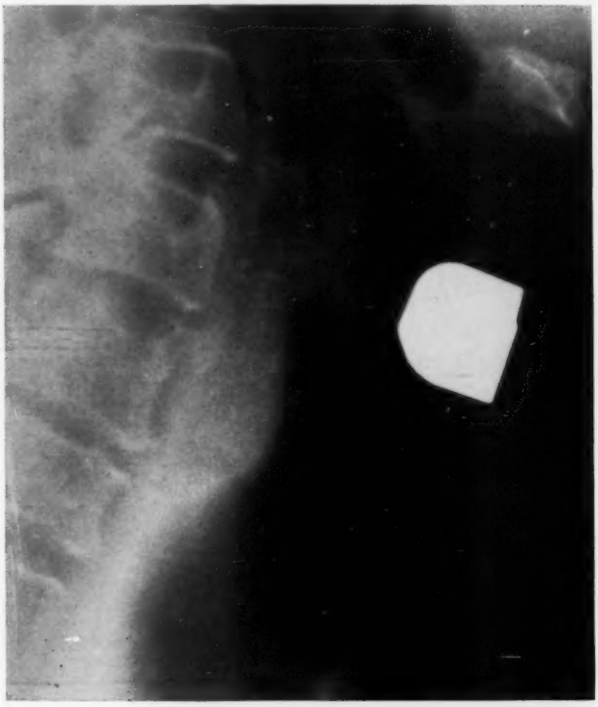

Fig. 3.

While an associate was observing the interior via direct laryngoscopy, a thin bladed sharp cataract knife was inserted through the cricothyroid membrane and brought up into the slot so as to divide the scar tissue in the midline. Care was taken not to injure the posterior wall and to bring the incision high enough to free the cord edges.

A plate of tantalum, 0.007 inches thick, was then placed through the saw cut in the cartilage like a center-board keel. It was trimmed so as to extend about half way back in the midline of the larynx, far enough to keep the raw surfaces apart but not so far as to interfere with the arytenoids. It reached from the cricothyroid membrane level to just above the upper surfaces of the cords. Posterior displacement was prevented by turning down tabs over each thyroid ala outside the perichondrium. The ribbon muscles were brought together in the midline over these tabs so as to prevent the plate from working outward. The fascia and skin were then closed without drains. Penicillin was given.

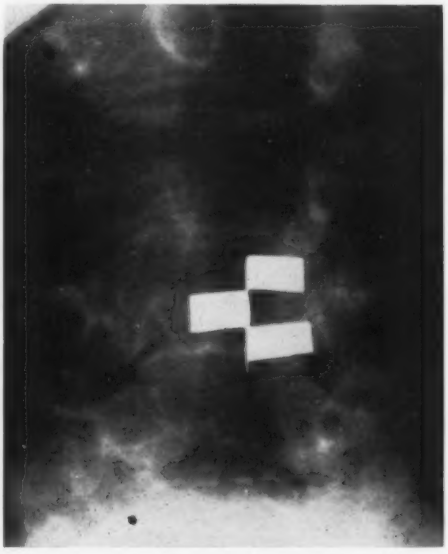

Fig. 4.

There was very little reaction. The patient swallowed normally the next day and left the hospital on the third day. For three days she inhaled penicillin dust t.i.d. and about this time was permitted to talk, since cough had not resulted in any subcutaneous emphysema. Her voice was better with the plate in than it had been before and breathing was very free. Fluoroscopy and films showed the plate to move with the larynx, not subject to external forces.

She returned to work. Frequent observation thereafter showed no inflammatory reaction to the presence of the tantalum at any time and it was judged after two months that epithelization was complete. She re-entered the hospital for removal of the plate. The incision was reopened and the plate removed without difficulty. There was no evident reaction outside the larynx. At this second hospital entry an attempt was made to take pictures of the plate *in situ* by direct laryngoscopy.

A recording was made of the patient's voice before the procedure was attempted, then during the time the tantalum plate was in place, and finally one 10 months after its removal.

The patient had promised to visit Dr. Paul Moses for voice training but so far has not done so. Abnormal habits of voice production had been necessary through the years of stenosis, and these have carried over to some extent. The larynx now looks so normal that I am sure better function is possible.

SUMMARY.

The problem of scar tissue web of the anterior third of the larynx is approached by applying the surgical principle of preventing healing from starting at the apex.

A case is described wherein a thin plate of tantalum was placed in the midsagittal plane of the larynx like a center-board keel, through a slot in the thyroid cartilage made via external incision. It caused little reaction and was left two months. Epithelization was complete at that time and the plate was removed by reopening the transverse neck incision.

A recording was made before, during and after the pressure, demonstrating improvement in voice.

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Note: These references comprise a mere sampling of the literature on the subject. Many of the more important articles are not mentioned. McHugh and Loch[18] cite 167 references to 1942.

THE LIMITS OF IMPROVEMENT OF HEARING FOLLOWING THE FENESTRATION OPERATION.*

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INTRODUCTION

In the fenestration operation for clinical otosclerosis a new pathway for the entrance of sound to the inner ear is established to take the place of the pathway through ossicles and oval window. The oval window has been closed more or less rigidly by the otosclerosis. As a part of the operation the ossicular chain is broken and the head of the malleus and the incus are removed. The drum membrane remains, covering the round window in a much-reduced middle ear. Sound reaches the fenestra (and round window) without benefit of the normal impedance-matching device of drum, ossicles and oval window. The theoretical (maximum) gain from such matching in transmitting sound from air to the fluid of the inner ear is about 30 db. Animal experiments[1] indicate a rise in threshold of about 28 db when the ossicular chain and the eardrum are removed (in the cat). *It is reasonable to expect, therefore, that after the best possible fenestration there will still be a loss of sensitivity of at least 20 db compared to normal.* The minimum residual loss may be less than 30 db if the normal benefit from drum and ossicles is less than the theoretical maximum of 30 db. The residual loss will be greater if the fenestra is not fully mobile, if there are cancellation effects between round window and oval window, or if the sensitivity of the inner ear is reduced for any reason.

*This work was begun under Contract N6onr-272 between the Office of Naval Research and Central Institute for the Deaf. It was continued under Contract V1001M-577 between Veterans Administration and Central Institute for the Deaf. The routine testing and the collection of data were transferred to the Hofheimer Audiology Laboratory at McMillan Hospital. The work there is supported by a grant from the Hofheimer Foundation.

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Several additional self-evident propositions are relevant to any test of our major hypothesis.

1) The standard error of measurement of the test for auditory acuity will contribute to the scatter of the results. The scatter introduced thereby will be in the shape of a normal distribution curve. [For Auditory Test No. 9 (recorded spondee words) the standard error of measurement obtained from tests on 55 patients in the present series is 3.1 db.][e]

2) "Normal" inner ears are not identical in sensitivity but will undoubtedly vary in random fashion within a few decibels of the average.

3) The acoustic efficiency of post-operative fenestras will also vary about a mean. This distribution will not necessarily be "normal" but may have a "tail" of relatively inefficient fenestras.

4) The sensitivity of the inner ears in patients with clinical otosclerosis will vary in the direction of impairment of hearing (nerve deafness) by more than the normal amount. High-tone nerve deafness is known to be present in the numerous cases that are clinically classified as "mixed deafness." In many of these cases a discrimination loss is demonstrated by the "PB" word lists.[3] The threshold for speech, as measured by Test No. 9, may be elevated by such nerve deafness. In fact, a nerve deafness involving frequencies 512 and 1024 need not necessarily cause a discrimination loss, providing the loss for the higher speech frequencies is not severe. Any such "nerve component" in the hearing loss for speech will obviously not be eliminated by the fenestration operation and will remain as an addition to the unavoidable conductive loss of 20 db due to absence of ossicles. The sum of these various factors will determine the minimum residual (post-operative) hearing loss for any particular case.

5) There is a minimum amount of conductive hearing loss that can possibly be produced by even complete

fixation of the stapes. Sound will be conducted to the inner ear by bone conduction and/or via the round window even if all movement of the stapes is abolished. From general acoustic experience in sound exclusion we may expect that the threshold for such alternative pathways of hearing will lie about 60 db above normal; *i.e.* at about the level of the phantom audiogram produced by an unmasked normal ear opposite a totally deaf ear under test. (Such a 60 db loss means that only one millionth of the normal amount of acoustic power reaches the inner ear.)

Our original hypotheses did not include the effect of fixation of the stapes on the bone-conduction threshold. This effect has been demonstrated recently by Carhart.[4] It is a conductive loss. Due to the increase in stiffness of the acoustic system in the *inner* ear when the oval window is rigidly closed by an immobile stapes, most sound waves are less effective in producing movement of endolymph regardless of how the sound waves are conducted to the inner ear. The "Carhart effect" appears as an elevation of the threshold for bone conduction, amounting to a maximum of 15 db at 2000 cps, but it is *not* due to nerve deafness. It is one of the factors that determines the exact value of the maximum conductive hearing loss that can be measured by air conduction. It is eliminated by the fenestration operation. If the residual cochlear function or the amount of "nerve impairment" is assessed by bone conduction, allowance must be made for this factor. As will be seen below we actually did allow for it in interpreting our own bone-conduction measurements, without specifically recognizing its existence, when we established our norms for bone conduction by averaging the *pre*-operative bone-conduction thresholds of the ears that showed the lowest *post*-operative thresholds by air conduction.

A corollary of our general hypothesis is that any hearing loss more than the maximum conductive hearing loss by air (not yet closely defined, but about 60 db) must include *1)* some nerve deafness and/or *2)* some *additional unusual* form of sound exclusion such as might be produced by rigid

closure of the round window. Neither of these two conditions will be alleviated or circumvented by fenestration; therefore, the *greatest possible gain for speech* that can be expected from fenestration is (maximum conductive loss) – (minimum unavoidable residual loss). Substituting the numerical values given above we have

$$\text{Maximum possible gain} = 60 - 20 = 40 \text{ db}$$

TESTS OF HEARING

We have tested our hypotheses by examining the results of 196 consecutive cases of fenestration performed by one of us (TEW) at McMillan Hospital (Washington University).

In none of the operations in question was a cartilage stopple employed, but nearly all of the cases operated by TEW without stopple (other than revisions) are included. No revisions of previous operations are included. The series begins arbitrarily in September, 1945, to include 81 eligible cases previously reported[2] plus 113 subsequent operations. From the 196 consecutive cases five are omitted for the following reasons:

Three patients from South America had more or less difficulty with English and their "thresholds for speech" are not comparable with the remainder of the group.

One patient left St. Louis before her post-operative hearing tests were completed.

One patient's fenestra closed very soon after the operation and before we had administered our usual speech tests. (Earlier post-operative pure-tone audiometry showed a satisfactory immediate result.)

Three patients gave very erratic and unreliable responses, both to pure-tone and to speech tests. This was noted at the time by our clinical assistants who administered the tests. Two of them are recognized as "psychopathic personalities." They are included in the overall count, but their data have been omitted from some of our graphs and calculations.

Loss for speech before and after operation was measured by Auditory Test No. 9 (recorded spondee words) administered at Central Institute for the Deaf or in the Hofheimer Audiology Laboratory at McMillan Hospital. The same recorded versions of the test were employed throughout, and physical and psycho-acoustic calibrations show the same normal threshold (22 db re 0.0002 microbar) for the two installations. Corrections were made for the slight differences in difficulty of individual lists as determined at the Psycho-Acoustic Laboratory.[5] The general arrangements and specifications of apparatus at Central Institute have been described elsewhere[6] and test facilities of equal or better quality and convenience have been installed in the Hofheimer Audiology Laboratory. The play-back characteristics at both laboratories conform to the specifications of the National Association of Broadcasters.[7]

Pure-tone audiometry by air conduction, and in most cases by bone conduction also, was carried out as part of the clinical routine at McMillan Hospital. These tests were less well standardized than the speech audiometry. The noise level in the audiometry booth was often above desirable limits. Before November 1947 several different audiometers were employed, and many different individuals have conducted the tests. The air-conduction data have been inspected carefully and they confirm the results of speech audiometry although they are less precise. The pre-operative pure-tone audiograms, in addition to the usual tuning fork tests, etc., formed part of the basis of diagnosis of the amount of nerve involvement; however, our graphs and calculations below are based entirely on the results of Auditory Test No. 9 except as specifically noted to the contrary.

This report deals entirely with "immediate" post-operative thresholds for speech. By "immediate" we mean not less than one and not more than four months after the operation. The test sessions were arranged to fall as close to three months after operation as could conveniently be arranged.

RESULTS

The *immediate post-operative losses for speech* are plotted in Fig. 1. The most recent 111 cases (December 1947 to July 1949 inclusive), not previously reported, are indicated by the solid blocks. The previous 80 cases (September 1945 through November 1947), on which a recent report[2] was based, are shown by the open blocks. The trends for the two parts of the series are quite similar, but both the median and the mode moved to slightly lower values in the more recent series.

The recent cases (solid) show a nearly "normal" distribution with median and mode both in the 26-27 db group. The "foot" of the curve includes two cases in the 18-19 db group, and there is a long "tail" of relatively unsuccessful cases extending to the right from 36 db. Nearly the same numerical values were obtained by estimating the loss for speech from the pure-tone audiogram (average for frequencies 500 and 1000 cps). The scatter of the thresholds for pure tone losses was somewhat greater, however, due to the larger standard error of measurement.[2]

For reference an arrow has been placed in Fig. 1 to indicate the generally accepted criterion of a successful result, namely a residual loss for speech of less than 30 db.

The actual distribution shown in Fig. 1. confirms empirically our proposition that *after the most successful fenestration there will remain a hearing loss for speech of about 20 db.*

We have not calculated the standard deviation for the distributions shown in Fig. 1 because the distributions are not normal in shape, but it is obvious that it is of the order of magnitude of $\pm$ 4 db if we ignore the "tail" beyond 36 db. This scatter of the nearly normal part of the distribution curve includes three elements enumerated above: *1)* the scatter due to the error of measurement inherent in the test itself, *2)* the scatter of sensitivities of the inner ears of the patients, and also *3)* the variations in actual acoustic conductivity of

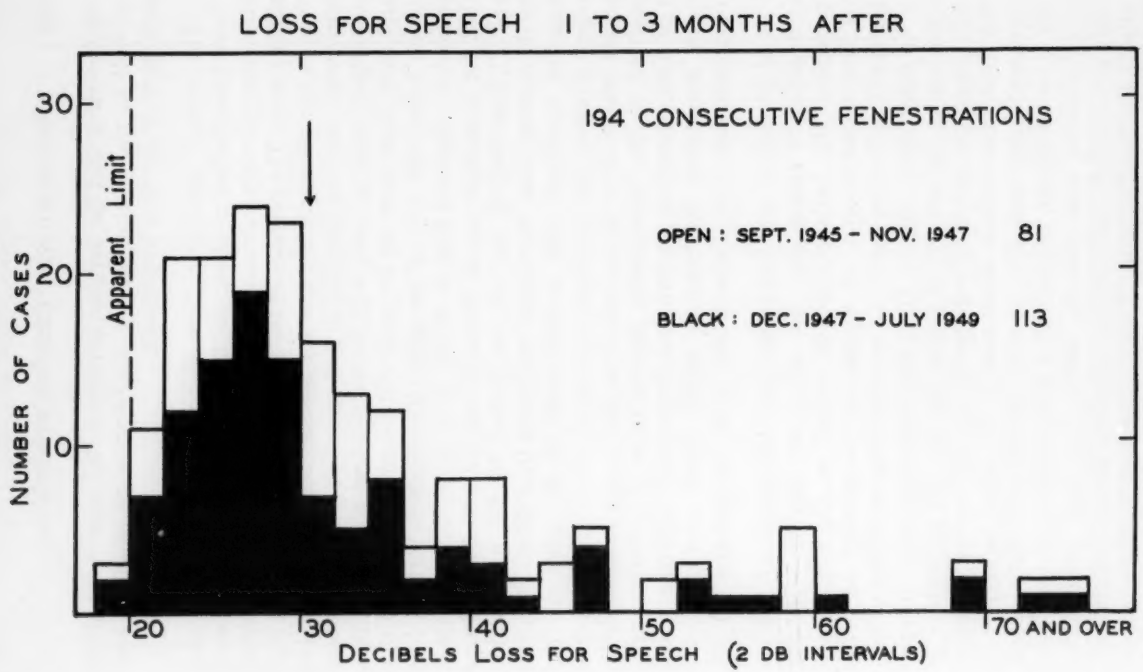

Fig. 1. Test results not corrected for slight differences in difficulty of the No. 9 lists. Two cases with a slight language difficulty, and three who gave erratic responses are included. One successful case operated late in July 1949 is omitted. (This chart was prepared for the meeting of the American Academy of Ophthalmology and Otolaryngology, October 1949, and is reproduced here without modification.)

the fenestras that the surgeon produced. We regard the shape as well as the position of the distribution as excellent confirmation of our first hypothesis.

The second hypothesis, that the maximum possible *gain* from fenestration is about 40 to 45 db, was tested by constructing the scattergram shown in Fig. 2. Each point represents a case. The position of the point on the vertical axis represents the pre-operative loss for speech, the position on the horizontal axis the immediate post-operative loss. Obviously cases that show no gain will lie on the heavy diagonal line, and cases that show 40 db gain will lie on the diagonal line 40 db below the line of no gain. The dashed vertical line indicates a residual loss of 20 db. It is immediately obvious that for cases with initial losses of less than 60 db the gains are all less than 40 db and are limited by the unavoidable 20 db residual loss. For cases with more than 60 db of initial loss the maximum gain is still about 40 db. (We use the round numbers for convenience because the scatter of a few points two or three db beyond these limits may reasonably be ascribed to the unavoidable uncertainties of measurement.) The data, therefore, confirm our hypotheses that the maximum conductive deafness is about 60 db and that the maximum gain from fenestration is 40 (or perhaps 43) db.

It is implicit in our hypotheses that the *degree of fixation of a patient's stapes does not affect his hearing after the operation.* His hearing depends instead on the function of his new fenestra, and, of course, on the amount of nerve deafness that he may have. To examine this point more closely we constructed Fig. 3. Here the cases have been grouped according to the severity of initial loss and the percentage of each group that achieved a given final result has been plotted. The final distributions show nearly the same form for all degrees of initial loss. For all of the groups with initial losses less than 60 db the chances of a good result (*i.e.* less than 30 db residual loss) are the same. For cases with initial losses greater than this the final distributions move systematically to the right. Figure 2 shows that nearly all the severe cases were actually diagnosed clinically as "mixed" deafness.

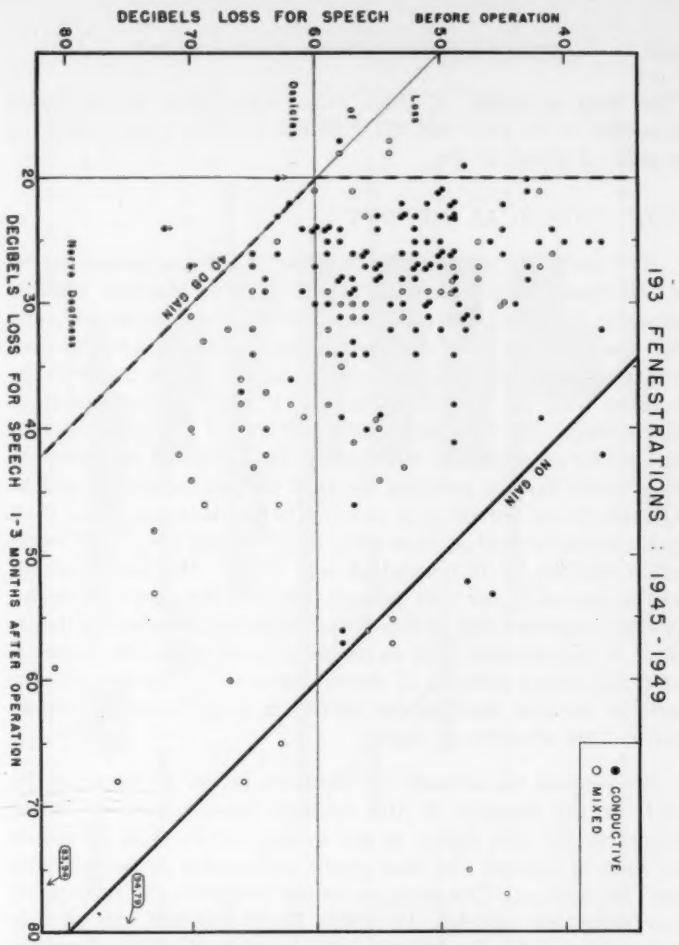

Fig. 2. Data corrected for differences in difficulty of the test lists. Solid dots represent pure conductive deafness. Open circles represent cases clinically diagnosed as mixed deafness before operation. Bottom-filled circles represent cases originally diagnosed as pure conductive but later proved to have significant nerve impairment.

Top-filled circle at 27, 70 is probably too low; the pre-operative threshold by Test No. 9 (70 db loss) is quite inconsistent with his performance on the PB-50 word lists and his pure-tone audiogram. The lowest solid dot (37, 66) is also probably too low; the pre-operative hearing loss for speech frequencies (pure-tone) was only 58 db. The point at 20, 63 is probably too low: pure-tone audiometry showed only 50 db pre-operative loss, and the performance on PB lists is most consistent with a loss of about 57 db. The bottom-filled circle at 24, 72 is certainly too low: another threshold test (for connected discourse), confirmed by the score on PB lists, gave a pre-operative loss of 62 db, not 72. These scattering points that seem to violate our generalizations appear on closer examination to be due to erratic performances on the threshold tests. Also most of the "conductive" cases that have an initial loss of more than 60 db show audiometric indications of a slight nerve involvement.

Two points fall beyond the limits of the chart. One case (79, 94) had very severe nerve impairment in addition to a conductive loss. Another, with very erratic pre-operative responses, is a complete failure with a post-operative loss of 94 db. The other erratic cases are plotted (68, 66 and 46, 63).

The final positions of these cases with large initial losses conform to the rule that the greatest possible improvement is a gain of about 40 db.

THE "SURGICAL DEFICIT"

The technical skill of the surgeon should be measured by *1)* the margin by which he fails to provide the best possible acoustic result, *2)* the permanence of the acoustic benefit, and *3)* freedom from undesirable clinical sequelae, such as discharging ears, etc. We are not concerned in the present discussion with the two latter points, in spite of their obvious importance. We do wish to point out that if a surgeon decides to operate on a patient with mixed deafness and achieves the best result that is possible for that patient he should not be blamed if the patient still has his nerve deafness. The total post-operative hearing loss after a technically successful operation may be 35 db instead of only 20 db. The social benefit of the operation for that patient may be very great, as shown by the improvement of his Social Adequacy Index for hearing.[8] If the surgeon fails to recognize in advance the presence of a significant amount of nerve deafness we should criticize not his surgical skill but his ability in diagnosis and prognosis, *i.e.* his *selection of cases.*

We propose the concept of "surgical deficit" to measure the skill of the surgeon in the acoustic plastic surgery called fenestration. His deficit is *the number of decibels by which he fails to achieve the best result reasonably to be expected for that patient.* The surgeon cannot perform the miracle of providing new ossicles. We allow 20 db residual loss on this account, even for the most favorable case. We also allow for any pre-existing hearing loss for speech *due to nerve deafness* that may have been present prior to operation.

Nerve deafness in the presence of conductive deafness may be difficult to measure. Nerve deafness for the lower speech frequencies is not necessarily revealed by a loss of discrimination. Our best present measure is the hearing loss by bone conduction for the speech frequencies. Unfortunately this

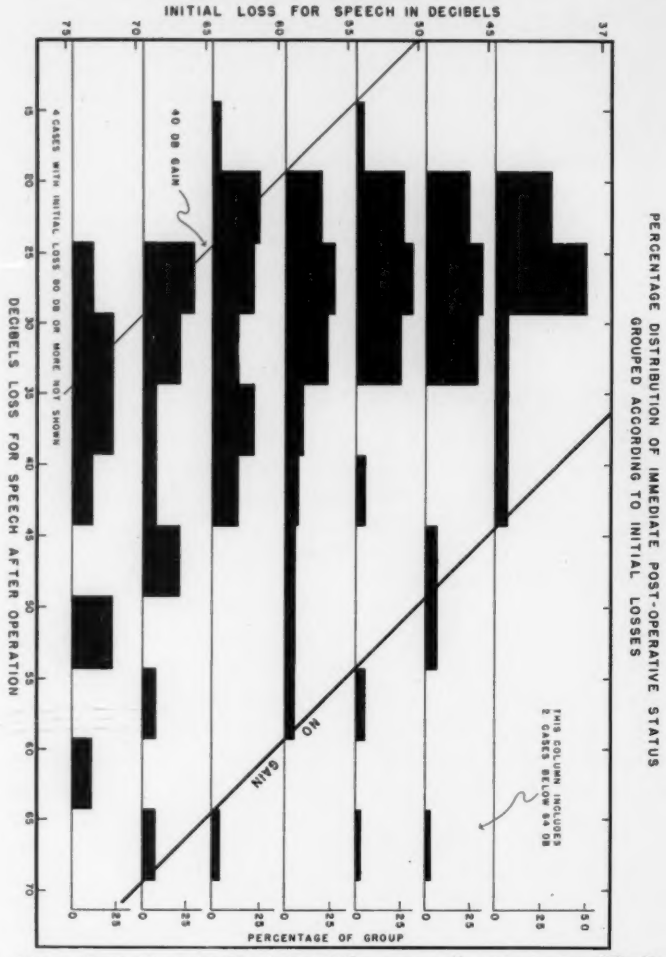

Fig. 3. Same data as in Fig. 2, but without correction to average difficulty. For these percentage distributions the corrections are immaterial. See text for further explanation.

measure is less precise than our measure of air-conduction loss for speech by Auditory Test No. 9. Reasons for the greater uncertainty include:

1) Faulty calibration of many bone-conduction receivers,

2) Possible masking by ambient noise: (Our pure-tone audiograms were taken during routine clinical testing, not in the Audiology Laboratory),

3) Uncertainty as to whether proper masking was used for the contralateral ear. (It may be *impossible* to adjust masking so as to measure the bone conduction threshold of an ear with mixed deafness if the opposite cochlea is normal but is occluded by otosclerosis.)

4) Elevation of the bone-conduction threshold by fixation of the stapes.[4] This elevation may amount to 8 or 10 db for the frequencies near 1000 cps that usually determine the thresholds for speech.

We attempted to allow for these factors by calculating the average pre-operative bone-conduction thresholds (average of 500 and 1000 cps) for the cases operated since October 1, 1947,* that attained the best post-operative results, *i.e.* within 25 db of normal. This group of ears cannot contain any with significant nerve impairment. The average pre-operative bone-conduction hearing loss for this group was 13 db. We considered 13 db apparent loss as "normal" when we estimated the presumable nerve impairment of other cases.

The best result that the surgeon can hope to achieve is to leave the patient with a loss for speech equal to 20 db (loss of ossicles) plus his nerve impairment. Residual loss for speech greater than this is the "surgical deficit." For our most successful cases the deficit is zero. The most common figure is a deficit of 6 or 7 db, both for cases without nerve impairment or with nerve impairment as great as 30 db. *A deficit of 12 db we consider as within reasonable limits.* The most common deficit of 6 or 7 db includes the normal variations in sensitivity of the inner ear, the errors of measurement, and the effects of uncontrollable minor variations in the physical properties of the tissues (such as thickness and stiffness of the skin) as well as factors under the control of the

*All of these cases were tested with the same audiometer, although by several different operators.

surgeon, such as size and placement of the fenestra and skin flaps, etc., etc. The greater the surgeon's skill (and good fortune) the smaller his deficit will be. At present we consider a deficit of 6 db a "good score." A residual hearing loss of 20 db (a deficit of zero) is a rare affair, like a sub-par "birdie" in golf. A deficit of 12 db is just passable. It lies close to the lower limit of our present "normal" distribution.

The complete formula for the surgical deficit is

$$\text{Deficit} = \text{(Residual air-conduction loss for speech)} - \text{(20 db)} - \text{(residual bone-conduction loss for speech.)}$$

The greatest uncertainty lies in the measurement of the final item.

We do not present in detail the data on surgical deficits. They involve corrections for nerve impairment, and the corrections calculated from the bone-conduction measurements are obviously excessive in several cases (presumably due to errors in the measurements) and lead to impossibly large negative deficits.

EXPLANATION OF SURGICAL FAILURES

The accumulation of cases at the left of the distribution in Figure 1 strongly suggests a "normal" distribution, *i.e.* cases that are fundamentally alike and differ only in random details that are not identified or under control of the surgeon.

In addition to this "normal" random scatter there is the long tail of unsuccessful cases from 35 db onward. Most, although not all, of those in the 32 to 40 db group gave evidence clinically and by bone-conduction audiometry of a mild nerve involvement (see Fig. 2). We have examined with special care, however, the cases with an immediate post-operative loss for speech of more than 40 db. These are failures, real or apparent, that lie entirely outside the range of the normal distribution. There are 30 such cases, 15.7 per cent of the series. Twenty-three of these "failures" had been recognized before operation as cases of "mixed" deafness.

The surgical deficits for 8 of the 23 "failures" with mixed deafness were not over 12 db. The surgeon did as good a job for them acoustically as he did for his average "successful" cases. In six more of these 23 cases the surgical deficit was from 13 to 20 db. These cases are not satisfactory, but neither are they very serious failures; and we should recall the uncertainties of our bone-conduction measurements. For the cases for which we have no bone-conduction measurements we cannot even estimate the surgical deficit, and for several others the measurements are open to more question as they were done before our present audiometer was installed.

Two of the remaining cases with large surgical deficits we may discount in part because in addition to their nerve impairment they gave erratic and unreliable responses during the hearing tests. The patients were characterized by the surgeon as "psychopathic personality." (Erratic responses and personality difficulties may have also contributed to the deficits in another case which is, however, fairly well explained by clearly recognized nerve deafness.)

In five cases there were large surgical deficits of 25 db or more, even when full allowance is made for a recognized and measured nerve deafness. For two of the five cases, which occurred very early (the second and the ninth) in the series as a whole, we have no explanation of the large surgical deficit other than the relative inexperience of the surgeon. In three of the remaining cases some surgical irregularity was noted that gives at least a plausible explanation. The commonest surgical accident (with or without nerve involvement) was a tendency to bleeding at the fenestra. The bleeding may have continued after the wound was closed. In two of the three present cases the fenestra had to be made through otosclerotic bone which bled profusely, and the third was noted at the time of operation for troublesome bone bleeding.

Four more of our cases with no nerve deafness but with large surgical deficits can be explained on purely surgical grounds. In one case the ear drum was torn and failed to heal. It is a matter of common experience that an intact ear

drum is essential for a satisfactory result, although the acoustic importance of the intact drum has never been adequately explained. In another case the horizontal canal of the membranous labyrinth was torn and a blood clot was seen in the vestibule. Curiously enough the loss for speech in this ear two months later was only 46 db, an 11 db gain from its pre-operative condition. This is a surgical deficit of only 26 db. The two other cases were noted at operation for troublesome bleeding at the fenestra. This is the commonest apparent cause of a large surgical deficit that we have been able to identify: five cases in all.

There remain only three cases of failure with large surgical deficits and one less severe case (surgical deficit of 20 db) for which we have no explanation. One of them, to be sure, was not a case of otosclerosis but of dense adhesive deafness. Two other cases of this type had good results, and we do not know why the fenestration should not have benefited this one also unless dense adhesions remained in the round window niche and somehow interfered with its function. The hearing loss was increased by the operation and the surgical deficit was 32 db. The other three, with surgical deficits of 37 and 27 and 21 db, were typical otosclerosis. Two were pure conductive deafness and the third had only moderate nerve impairment. All three were "perfect operations." Their fenestras are open but their hearing in each case is within 10 db of their pre-operative levels. In these cases it is legitimate to speculate as to the probability of closure of the round window by the otosclerotic process. It is important, however, that *in only three cases (1.6 per cent) in the series is it really legitimate to postulate some such additional purely hypothetical factor.* In all other cases we have at least a plausible and in many cases a quantitative explanation of the immediate post-operative status, whether the result is clinically satisfactory or not and whether the "surgical deficit" is large or small. Only two or three more cases at most could reasonably be added to the "unexplained" list as being insufficiently explained on the basis of nerve impairment.

THE SURGEON'S SCORE

The evaluation of a surgeon as a fenestrator is apparently two-fold: first, what are the chances of a serious surgical failure, *i.e.* a surgical deficit of 20 db or more, due to bleeding, torn labyrinth, torn drum, etc.? For the present the surgeon must also carry the responsibility for the X factor also, whether it is an undiagnosed fixation of the round window or whatever else it may be. Second, what is his mean surgical deficit for cases that are not failures and what is his standard deviation? In other words, what is his expected performance, and what are the chances of any particular case coming within say 5 db of the average? Third (not considered in this paper), what are the chances that improvement will be permanent? Fourth, how good are the surgeon's prognoses? Does he assess accurately the presence and importance of nerve impairment and discrimination loss? These conditions he cannot be expected to alleviate, but he must evaluate them accurately and not mislead himself or his patients as to the probable extent of the benefit of the operation. (It is here that the Social Adequacy Index is helpful.)

We may reckon one kind of rating for a surgeon by noting the incidence of "failures" for which he is clearly to blame or for which no reasonable cause such as nerve involvement, language difficulty, etc., can be found. For example, let us consider a surgical deficit of 20 to 25 db as a simple failure and over 25 db as a serious failure. The 30 cases in the present series with post-operative losses of more than 40 db, discussed above, were grouped in this way. Twelve of them prove to be satisfactory surgical results when allowance is made for nerve impairment, etc., and five more are failures but not serious failures. Thirteen, however, or 6.8 per cent or one in 15, are serious failures.

The incidence of these serious failures in our series is illuminating, however. Six of the 13 occurred in the first 29 cases of the series, between September 28, 1945, and November 1, 1946. One cannot escape the conclusion that, although the surgeon had performed 53 fenestration operations (most

of them using the cartilage stopple), and several revisions, before September 28, 1945, his technique was not fully developed until early November 1946. By this time he had performed 165 fenestrations, not counting revisions. Since November 1, 1946, his incidence of serious failures as defined above has been 7 out of 167, or 4.2 per cent.

SELECTION OF CASES

The incidence of cases with severe nerve involvement was much higher in 1945 and 1946 than it has been since. The surgeon has, since 1946 when we developed the concept of the Social Adequacy Index, discouraged from operation more and more strongly all patients with evidence of serious nerve involvement. The nerve involvement is often shown very clearly by a severe loss for high tones by bone conduction and a significant discrimination loss on our monosyllabic word test. Partly as a result of the improved selection cases there are only four cases since December 10, 1948 (the last 52 cases), with post-operative hearing losses as much as 40 db. All four were operated in February, 1949. Three of the four had considerable nerve involvement, and no cases with so much nerve loss have been operated on since.

The importance of selection of cases is shown in Figure 4. Here we have plotted separately the immediate post-operative results for the cases diagnosed clinically before operation as "pure conductive deafness" and as "mixed deafness" respectively. Two cases discovered after operation to have a very significant nerve loss have been transferred from the pure conductive to the mixed category. As before, the solid blocks represent the cases operated since November 30, 1947, and the open blocks those prior to that date.

No attempt is made in this figure to correct for the amount of nerve deafness in the mixed cases. In many of them the nerve loss affected only the high tones and did not elevate the threshold for the lower speech frequencies. That is why some of the most successful cases are in the mixed group. In fact the mixed group is distributed almost evenly from 17 db

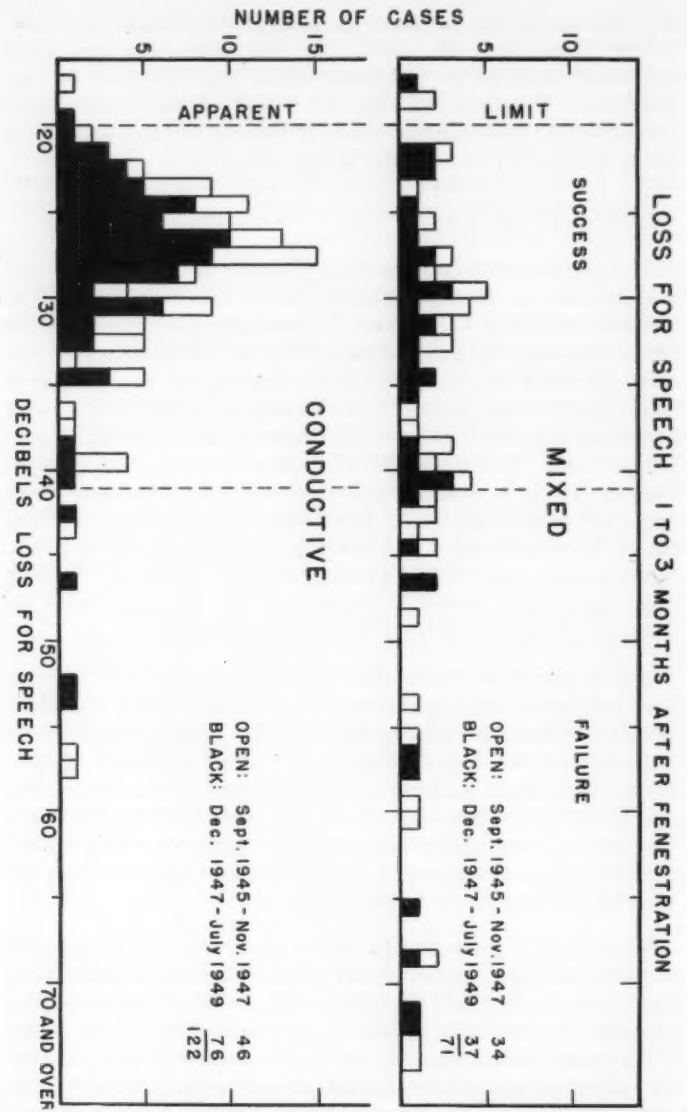

Fig. 4. Test results have here been corrected to average difficulty. Cases are subdivided into conductive and mixed deafness. All three cases with language difficulty are excluded, but the three who gave erratic responses (all with mixed deafness) are retained.

residual loss to complete failure (over 70 db residual loss); but among the 15 cases identified by the surgeon as having the greatest nerve involvement only one had a post-operative loss as small as 35 db.

The 77 more recent cases of pure conductive deafness form distribution centering at 26 db. If we disregard the "tail," *i.e.* cases with more than 35 db loss, the remaining 69 cases form an almost perfect normal distribution with a mean of 26.4 db and a standard deviation just over 3.3 db. These two numbers and his incidence of failures measure the surgeon's skill in providing the best possible immediate acoustic result.

The 46 earlier cases of pure conductive deafness include a "tail" of eight cases with residual losses of more than 35 db. The percentage of such unsuccessful cases is 17 per cent as compared with 9.2 per cent in the recent series. The mean for the remaining 38 cases is 27.1 db with a standard deviation of about 3.8 db. In the more recent series the surgeon averaged a fraction of a decibel better and became a little more consistent in his performance. This is just the sort of improvement that might well be the result of accumulated experience and practice.

The trend of the results with the pure conductive deafness is shown graphically in Fig. 5. Here the results have been averaged in groups of 10 to 15, omitting the failures with post-operative losses of more than 40 db. The size of the groups was chosen arbitrarily to bring the divisions at the beginning of calendar years, vacations, etc. (The "recent series" is the cases operated in 1948 and 1949 plus 2 in December 1947.) There is an abrupt improvement in mean score after the summer of 1947. The fluctuations since that time are not significant, and depend chiefly on whether one, two or three of the less successful cases (30 to 40 db residual loss) happen to fall in a particular period. The general average for 1948 is 26.5 db; for 1949 it is 26.6 db. There is no evidence here that improvement in the surgeon's skill still continues. On the other hand there have been no serious

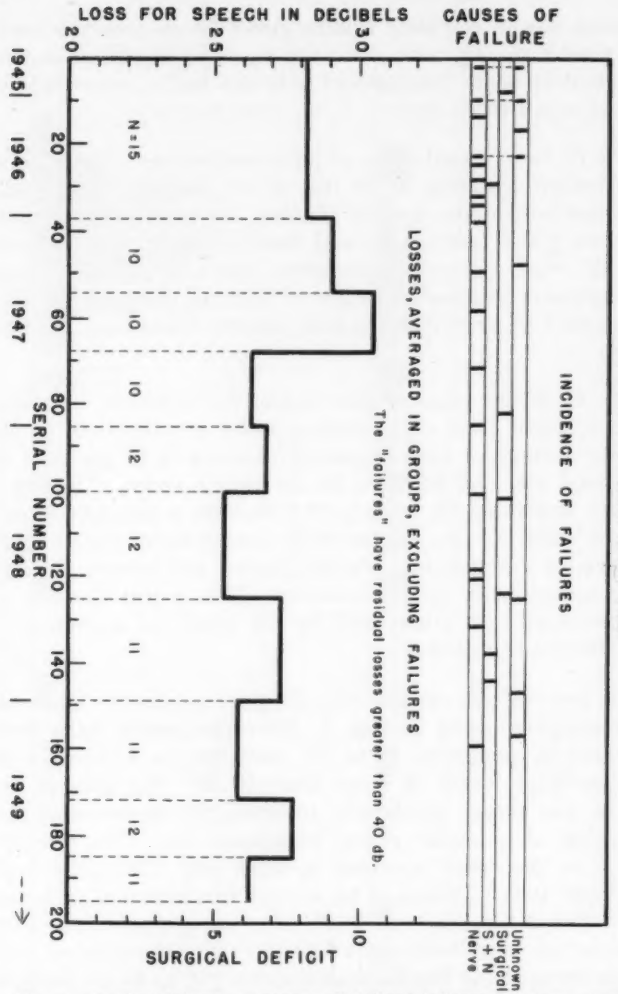

Fig. 5. The breakdown of causes of failure follows the text. "Failure" means an immediate post-operative hearing loss for speech of more than 40 db, and includes cases of nerve deafness, surgical accident and unknown causes. One case (with a "psycopathic personality") whose responses were unreliable has been omitted. Two other erratic cases, in which nerve deafness was obvious and severe, have been included. As far as possible each case has been classed in the single category that seems to be the chief cause of the failure, but six are divided between two categories. See text for explanation of average losses of the successful cases of conductive deafness.

failures since February 1949. Experience and good selection of cases probably account largely for this; but even with the greatest skill and care we must expect sooner or later to encounter more failures from the "unknown" causes. It is evident, however, that the immediate acoustic result for future members of this series can be predicted with considerable assurance and fair accuracy. Indeed, recently the surgeon has been in the habit of indicating on his patient's chart the expected S.A.I. He has been consistently accurate to with within ± 5 S.A.I. points. The expectations for the permanence of their gains we shall consider at a later time when more long-term data are available.

SUMMARY.

The hearing losses for speech have been measured before and shortly (about three months) after 195 consecutive fenestration operations.

The data are compared with the results of an analysis of known physical and psycho-acoustic factors involved. They are in excellent agreement, and support the following propositions:

1) The best result to be expected following fenestration is a residual hearing loss for speech of 20 db. This minimum loss depends primarily on absence of the ossicles.

2) The residual hearing losses of successful cases scatter at random about a mean. The scatter is due to

  a) The "error of measurement" of the hearing test:

  b) Normal variations in the acuity of the inner ear;

  c) Variations in the physical properties of the patients' skin flaps and other tissues, and uncontrolled minor variations in surgical procedure;

3) The maximum possible *conductive* hearing loss for speech is about 60 db. When speech is more than 60 db above normal threshold it can be heard by bone

conduction. Losses greater than 60 db therefore mean that more or less nerve deafness must be present. This is confirmed by bone-conduction audiometry.

4) Nerve deafness is not alleviated by the fenestration operation. The *maximum gain* that can be expected from fenestration is therefore 60 db – 20 db, *i.e.* 40 db.

5) The residual hearing to be expected after fenestration depends on the amount and character of *nerve* impairment (if any) but not on the amount of pre-operative *conductive* deafness.

The number of decibels by which a patient's immediate post-operative hearing loss for speech exceeds the unavoidable minimum we call the "surgical deficit." For pure conduction deafness the deficit is the excess above 20 db residual loss. For mixed deafness additional allowance is made for the "nerve" component of the deafness. The latter is at present best measured as the hearing loss by bone conduction. Several difficulties attending such measurements are discussed.

The mean post-operative hearing loss for our 69 cases of pure conductive deafness operated since December 1, 1947, excluding definitely unsatisfactory cases with final losses greater than 35 db, is 26.4 $\pm$ 3.3 db.

The incidence of failures, the mean surgical deficit for the remaining cases, and its standard deviation are three appropriate measures (among others) for evaluating a surgeon's technical proficiency. If accurate estimates of nerve impairment are available we can, from these measures of the surgeon's skill, make prognoses of known probability. Thirty (15.7 per cent) of our series had an immediate post-operative loss for speech of more than 40 db. Fourteen of these "failures" are due largely or wholly to the presence of significant nerve impairment.

Twelve failures (6.3 per cent of the series) are due to known or probable surgical "accidents," usually bleeding into the fenestra as from otosclerotic bone. Only three failures (1.6 per cent of the series) are completely without explanation.

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A STUDY OF DELAYED BLEEDING FOLLOWING REMOVAL OF THE TONSILS AND ADENOIDS.*†

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Hemorrhages following the removal of the tonsils and adenoids are often embarrassing to the otolaryngologist and may even be fraught with grave consequences. Anything that may be done to lessen the occurrence or alleviate the severity of delayed bleeding will ease the physician's task, lighten his responsibility and enhance his reputation in the sight of his patients. A study of the causes of postoperative bleeding was undertaken in the hope that something worthwhile might be discovered.

Statistics showing the incidence of hemorrhages varied considerably among equally competent surgeons. A review of the author's own records indicated that apparently a record was made on a patient's chart only in the more or less severe cases. No report of slight bleeding was found, although it was easy to recall that such cases had occurred. Fox and West[1] have stressed the importance of persistent follow-up reports on all cases, even using the telephone and mail service when necessary, otherwise many cases of bleeding will be missed. Due to such previous laxity, it was impossible to establish an accurate control series for comparison with the experimental series that are to follow. Possibly some authors distinguish between hemorrhages and slight to moderate bleeding which causes little inconvenience and stops spontaneously within a few minutes. In this case such statistics would indicate more favorable results.

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HISTORICAL.

Since 1940, a number of articles have appeared in the literature on the relationship of hypoprothrombinemia and the salicylates, more particularly acetylsalicylic acid. Attempts to correct this deficiency by the administration of vitamin K have been reported both favorably and unfavorably by numerous investigators. In this year, Elliott, Isaacs and Ivy[2] produced a dietary prothrombin deficiency in the rat and showed that a complete return to normal could be obtained by the administration of vitamin K. In 1941, Kark and Souter[3] reported that spontaneous bleeding might occur from pathological surfaces, such as wounds, ulcers, etc., when the prothrombin level in the blood is down to 35 per cent and a hemorrhagic diathesis may occur if it is lowered to 15 to 20 per cent. They further state that the intravenous administration of vitamin K "controls hemorrhage from hypoprothrombinemia in one and one-half to three hours while the blood prothrombin level rises rapidly and reaches a normal level within 24 to 48 hours." Other authors, especially Link[4] and his co-workers, have demonstrated a parallelism between the hypoprothrombinemia produced by hemorrhagic sweet clover disease and salicylic acid. Shapiro, Redish and Campbell[5] state that "the action of salicylate is apparently identical with that of the anticoagulant, dicumarol, but less effective," and that acetylsalicylic acid is more potent in this respect than sodium salicylate.

Clinical reports have been appearing since then showing a close correlation between the use of salicylates and bleeding that occurs after the removal of tonsils and adenoids. Among the first of these was an article by Singer[6] that pointed out the greater frequency of postoperative hemorrhages in this country compared to European countries was due to the prevailing custom of using aspirin here compared to aminopyrine over there. In the salicylate medication of rheumatic fever patients, Rapoport, Wing and Guest[7] state that there is a "marked prolongation of the prothrombin time of the blood." Meyer and Howard[8] confirmed this and showed that the concomitant administration of vitamin K prevented the develop-

ment of a hypoprothrombinemia. Fox and West,[1] as well as others (Stone[9] and Howell[10]), have shown that vitamin K is obtained physiologically from the normal intestinal flora and stored in the liver. The absence of hepatic disease is, therefore, a primary consideration in all of the reports on salicylate induced hypoprothrombinemia. Other reports advising the simultaneous use of vitamin K with acetylsalicylic acid have shown marked improvement in the reduction of postoperative hemorrhages. Fox and West[1] have attacked the accuracy of some of the reports on the basis of "too little laboratory data." They have concluded that there is no protective value in using vitamin K postoperatively as hypoprothrombinemia is not the cause of delayed tonsillar hemorrhage. They feel that the high incidence of bleeding following the use of chewing gum containing aceylsalicylic acid is due probably to some local effect on the wound. Ersner[11] and associates, in 1934, indicated that saliva mixed with blood becomes a potent factor in decreasing the coagulation time and that the presence of aspirin in the saliva delayed the formation of a clot.

The rôle played by vitamin C as well as vitamin K in preventing hypoprothrombinemia has been stressed by Neivert[12,13] and others.

PHYSIOLOGY.

The classical theory of blood coagulation established in the latter part of the last century is still generally accepted among most investigators. Prothrombin, thromboplastin and calcium unite to form thrombin. Thrombin and fibrinogen then react to produce fibrin or the clot. Anything that will lower the prothrombin content of the blood plasma thus will interfere with the normal process of blood coagulation. It is with this phase that the otolaryngologist is most concerned as a means of preventing delayed postoperative bleeding.

CLINICAL PROCEDURES.

In order to make an accurate study of postoperative bleeding, every case was operated upon by the author and followed

at least 10 days by routine postoperative office call or by a report over the telephone. All children were done under general anesthesia and in most cases both the tonsils and adenoids were removed, although in some only one or the other was done. The tables do not differentiate in this matter. The adults were done under local anesthesia, employing 1 per cent procaine hydrochloride with eight drops of epinephrine hydrochloride to the ounce for better hemostasis. In removing the adenoids, a LaForce adenotome was used first in the midline and then again on each side, being careful to keep the blade from injuring the cushion of the Eustachian tubes. The area was inspected and any residual lymphoid tissue removed with a punch forceps. The tonsils were removed by blunt dissection and snare, sharp dissection being employed only in the case of marked fibrosis of the capsule. In general, all primary bleeding was controlled by sponge pressure, but catgut sutures or ligatures were used whenever necessary.

For the purposes of this paper only delayed bleeding is considered, that is, bleeding which occurred only on the second postoperative day or later. Also, all cases of bleeding, no matter how slight, are recorded, and on this account the term "hemorrhage" will not be used except in case of a direct quotation where the patient used the term in his report.

In the first series, comprising 164 cases, 10 mg. Synkayvite were given with meals three times daily and one tablet of Aspergum was ordered about 10 minutes before eating. In this series there were 12 cases (7.31 per cent) of bleeding, only five requiring medical attention (3.05 per cent) (see Chart 1).

In the second series, comprising 310 cases, 10 mg. Synkayvite, 250 mg. ascorbic acid and one tablet of Aspergum were given three times daily with meals. In this series there were 32 cases of bleeding (10.32 per cent) but only three that required medical attention (0.97 per cent) (see Chart 2).

In the third series comprising 179 cases there were 25 cases of bleeding (13.96 per cent), of which 13 (7.26 per cent) required medical attention (see Chart 3).

CHART 1.
SYNKAYVITE AND ASPERGUM.

No.	Name	Age	Date Oper.	Postoper. Day of Bleeding	Remarks
1	E. P.	5	9/21/45	5th	Bled from nose freely.
2	J. R.	8	10/12/45	5th	Profuse adenoid bleeding; postnasal tampon used.
3	F. K.	6	2/16/46	6th	Moderate bleeding
4	K. K.	32	2/18/46	6th	Moderate bleeding off and on; small clot removed on the 8th day.
5	E. B.	38	4/ 1/46	5th	Slight bleeding.
6	L. M.	5	5/ 9/46	8th	"Had a gush of blood from the nose lasting one minute. Many nosebleeds all of life."
7	S. S.	5	5/ 9/46	7th	Adenoid bleeding rather severe.
8	B. W.	11	5/14/46	6th	Slight spotting on pillow; did not awaken.
9	J. O.	6	7/ 1/46	7th	Very slight bleeding; "like a tooth pulled."
10	A. G.	5	7/ 5/46	8th	Adenoid bleeding; sponge pressure used.
11	G. S.	27	7/ 9/46	6th	Bleeding tonsil fossa; sponge pressure used.
12	D. M.	8	7/11/46	7th	Severe adenoid bleeding; postnasal tampon used.

Total number of cases in this series was 164. Ten mg. Synkayvite were given three times a day and Aspergum before meals. Cases requiring medical care were five. Cases stopping spontaneously were seven.

CHART 2.
SYNKAYVITE, ASCORBIC ACID AND ASPERGUM.

No.	Name	Age	Date Oper.	Postoper. Day of Bleeding	Remarks
1	E. K.	29	11/15/46	5th	Reported as "slight hemorrhage."
2	J. C.	4	11/18/46	7th	Severe adenoid bleeding; sponge pressure used; took only two doses of medication.
3	D. G.	4	11/22/46	3rd	Moderate adenoid bleeding.
4	N. K.	14	11/26/46	5th	Slight spotting.
5	B. B.	7	11/26/46	5th	Slight adenoid bleeding.
6	J. R.	4	1/14/47	4th	"Slight bleeding. Lasted only a few minutes."
7	T. F.	3	4/21/47	9th	Adenoid bleeding; postnasal tampon used.
8	N. K.	4	5/ 5/47	3rd	Adenoid bleeding.
9	P. O.	10	5/10/47	5th	Moderate bleeding.
10	A. K.	8	6/ 6/47	3rd	Very slight bleeding.
11	D. C.	5	6/17/47	4th	Slight adenoid bleeding.
12	D. S.	4	6/27/47	8th	Slight adenoid bleeding.
13	J. T.	4	7/11/47	5th	Slight bleeding.
14	R. K.	18	8/25/47	6th	Bleeding tonsil fossa; sponge pressure used.
15	I. P.	5	10/ 6/47	6th	Moderate bleeding from nose.
16	R. F.	6	10/24/47	8th	Slight adenoid bleeding.
17	M. M.	4	10/31/47	11th	Slight bleeding.
18	M. J.	4	11/ 4/47	8th	"Expectorated small clot from the throat."
19	R. B.	3	11/17/47	5th	"Expectorated a large clot from the throat."
20	R. B.	6	11/24/47	6th	Bleeding from nose.
21	W. H.	4	11/28/47	5th	Slight bleeding.
22	B. S.	11	12/ 5/47	5th	Slight adenoid bleeding.
23	D. B.	7	12/15/47	6th	Moderately severe bleeding.
24	B. B.	7	12/29/47	8th	Adenoid bleeding.
25	K. C.	5	1/16/48	9th	Slight adenoid bleeding.
26	J. S.	5	1/17/48	4th	Moderate adenoid bleeding.
27	J. G.	5	3/ 5/48	7th	Moderately severe adenoid bleeding.
28	J. C.	7	3/26/48	4th	Moderately severe adenoid bleeding.
29	S. M.	4	4/30/48	6th	"About a teaspoonful from the nose."
30	T. H.	5	6/24/48	5th	"Slight bleeding from the nose for about two minutes."
31	J. S.	5	7/ 1/48	6th	Slight bleeding off and on seventh and eighth days.
32	M. D.	6	7/16/48	7th	Slight bleeding from the nose.

There was a total of 310 cases in this series. All had 10 mg. Synkayvite three times a day, 250 mg. ascorbic acid three times a day and Aspergum before meals.

Cases requiring medical attention were three, the other 29 stopped spontaneously.

CHART 3.
ASCORBIC ACID AND ASPERGUM.

No.	Name	Age	Date Oper.	Postoper. Day of Bleeding	Remarks
1	B. J.	5	9/17/48	7th	Severe adenoid bleeding; postnasal tampon and three transfusions.
2	C. P.	6	10/29/48	2nd	Severe adenoid bleeding; postnasal tampon.
3	S. K.	7	11/ 1/48	3rd-8th	Moderate adenoid bleeding.
4	A. Q.	4	11/ 3/48	9th	Severe adenoid bleeding; postnasal tampon.
5	J. G.	4	11/26/48	5th	"Very slight bleeding for a few seconds."
6	B. D.	6	1/10/49	8th	"Bled about a teaspoonful."
7	P. B.	6	1/21/49	6th	Small amount of bloody emesis.
8	K. P.	5	2/11/49	6th	Slight bleeding.
9	C. K.	6	3/ 1/49	8th	Severe adenoid bleeding; postnasal tampon.
10	C. S.	12	3/28/49	11th	Severe adenoid bleeding; postnasal tampon.
11	T. S.	10	4/12/49	6th	"Slight nosebleed."
12	H. S.	4	4/20/49	9th	Slight spotting on the pillow.
13	E. R.	3	5/ 3/49	3rd	"Slight nosebleed."
14	J. L.	6	5/ 5/49	3rd	Bleeding tonsil fossa; sponge pressure.
15	M. K.	4	5/16/49	7th	Severe adenoid bleeding; sponge pressure.
16	K. R.	9	5/20/49	4th-6th	Moderate bleeding tonsil; two days later adenoids.
17	L. H.	5	5/27/49	7th	Severe adenoid bleeding; postnasal tampon.
18	W. S.	5	6/17/49	4th	Adenoid bleeding; sponge pressure.
19	A. D.	7	6/17/49	6th	Slight adenoid bleeding.
20	J. W.	3	6/24/49	6th	Severe adenoid bleeding; postnasal tampon.
21	M. S.	4	7/ 6/49	6th	Slight spotting from nose.
22	K. Y.	8	7/ 8/49	5th	Moderately severe adenoid bleeding; sponge pressure.
23	P. M.	8	7/ 8/49	8th	Moderately bleeding tonsil fossa; sponge pressure.
24	W. H.	6	7/18/49	3rd	Adenoid bleeding about 10 minutes.
25	L. M.	11	7/22/49	6th	Moderate adenoid bleeding; sponge pressure.

There were a total of 179 cases in this series. Thirteen required medical attention; 12 cases stopped spontaneously. All were given 250 mg. ascorbic acid three times daily and Aspergum before meals.

COMMENT.

The one factor common to each of the series was the use of Aspergum (chewing gum with approximately 3.5 gr. acetylsalicylic acid). Livingston and Neary,[14] in a small series of cases, were unable to find any evidence that this would produce a hypoprothrombinemia. Fox and West[1] are inclined to agree with this and think that late tonsillar bleeding is due to some local effect of the aspirin on the wound. A contrary view is expressed by Neivert[12] when he says "most assuredly such a rise (elevation of prothrombin time) will occur eventually," as shown by his experimental studies on normal individuals.

The synthetic vitamin K used is commonly known as Synkayvite and has the formula 2-methyl-1,4-naphthaquinone. It was given in the first and second series to see if it might materially reduce the incidence of postoperative bleeding. As mentioned previously, no attempt had been made to keep as accurate a record of bleeding previously to this study when acetylsalicylic acid was the only form of medication employed, so no true control can be established. The fact that bleeding did occur seriously enough in 3.05 per cent of the cases to require medical attention in order to control it indicates that most certainly vitamin K by itself will not prevent delayed secondary bleeding.

In the second series of cases ascorbic acid was given in conjunction with the vitamin K. It is well known that ascorbic acid is beneficial in most body healing processes. Where a patient is so severely handicapped in the normal ingestion of food, as in the case of a tonsillectomy, the nutrient value of ascorbic acid on the granulating surface might be important in reducing the incidence of bleeding. Although the overall occurrence was higher than with vitamin K alone, there were many cases of slight bleeding, so that the number requiring medical attention was just under 1 per cent. Thus, it would seem reasonable to advise the use of ascorbic acid in conjunction with vitamin K as a probable beneficial measure in cases of tonsillectomy.

In the third series, ascorbic acid and Aspergum alone were used. The overall bleeding was highest in this group, 13.96 per cent, and the serious ones requiring medical attention amounted to 7.26 per cent. By comparing this group with the first, then, it is possible to make a reasonable conclusion that the use of vitamin K reduces the overall percentage of bleeding from 13.96 to 7.31 and the serious ones from 7.26 to 3.05. Since the use of Aspergum was common to all three series, any local action on the wound surfaces that would predispose to bleeding would be nullified for statistical purposes and would not be an important factor. Of greater importance in this clinical study is the effect of vitamin K and more particularly the simultaneous administration of vitamin K and ascorbic acid.

A study of Chart 4 shows a compilation of the days during which postoperative bleeding occurred. It is readily seen that the critical period lies between the third and eighth days,

CHART 4.

Day of Post-operative Bleeding	Number of Cases
2nd	1
3rd	6
4th	5
5th	14
6th	17
7th	10
8th	11
9th	4
10th	0
11th	2

In the above computation one case bled on the third and eighth days, another on the fourth and sixth days, and a third on the sixth, seventh and eighth days and are listed accordingly in the second column.

although it may take place at any time through the eleventh day, the greatest frequency being on the fifth and sixth days. Three cases bled on more than one day. It is during this

period that the granulation tissue is forming between the raw surface and the exudative membrane that forms over it. As this membrane its gradually displaced and sloughs away a capillary bed or an individual vessel may become exposed and eroded, thereby starting the bleeding.

Predisposing factors that may increase the incidence of bleeding are the usual blood dyscrasias, congestion of granulations due to the presence of infections or incompletely resolved recent acute exacerbations of respiratory illnesses, dietary deficiencies, particularly the proteins and vitamins, hypertension and local trauma to the tissues, such as might be caused by certain types of food. Any case of suspected blood dyscrasia should be carefully checked by appropriate blood studies, not depending upon the results of the usual coagulation or bleeding time which are taken routinely. The determination of the prothrombin time will afford more accurate information and should be done whenever possible in the preoperative examination of the patient. The operation should never be done in the presence of an acute infection or even in a recent one unless adequate time has been allowed for complete resolution to take place. In questionable cases chemotherapy or the administration of certain of the antibiotics may be given preoperatively and for a day or two postoperatively. McGovern[15] recommends the use of sulfathiazole chewing gum, while Fox and West[1] advocate the use of lozenges containing benzocain and tyrothricin — tyrozets — for their local action in keeping down infection and the latter also for relieving discomfort. These might well be used in tonsillectomies for this purpose, but it is difficult to see how they would be of any benefit as far as the adenoids are concerned. In the author's series covering 653 cases the incidence of bleeding from the adenoids was far greater than from the tonsil fossae.

CONCLUSIONS.

The preponderance of evidence in the literature indicates that salicylates have a depressant action on the formation of prothrombin and thus predispose to postoperative bleeding.

Synkayvite given postoperatively exerts a beneficial effect in lowering the incidence of bleeding.

Synkayvite and ascorbic acid given simultaneously exert a still greater effect in reducing this incidence.

The critical period for bleeding to occur ranges from the third to the ninth days, with most occurring on the fifth and sixth days.

Careful preoperative examination including appropriate blood studies and choosing a time free from recent infections are other important factors in selecting cases for operation.

SUMMARY.

A report of 653 consecutive cases of tonsillectomy and adenoidectomy was made to study the incidence of postoperative bleeding.

Three series of investigations were made to determine if acetylsalicylic acid, Synkayvite and ascorbic acid might have a bearing on the subject.

A study of the time for the occurrence of postoperative bleeding was made.

30 North Michigan Avenue.

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BOOK REVIEW

Bronchography. By Eelco Huizinga, M.D., and G. J. Smelt, M.D., Department of Oto-Rhino-Laryngology, University of Groningen, 270 pages with 146 illustrations. Assen, Netherlands: Van Gorcum & Co., Ltd., 1950. Price: Hfl 39 (Dutch currency; about $10.00 in American funds).

This is an excellent and detailed story of bronchography. The authors devote the first part of this book to the anatomy and physiology of the bronchial tree. They treat the subject comprehensively.

General considerations of bronchography with indications and contraindications compose Chapter 3, and a valuable discussion of the Roentgen anatomy of the normal bronchogram follows.

The discussion of the interpretation of bronchograms with cases and many excellent illustrations is very valuable.

The last chapter is a detailed description of the authors' technique in bronchoscopy and the bibliography is complete.

A very valuable book for any who are interested in diseases of the chest and in endoscopy. T. E. W.

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The next meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Illinois Hotel, Bloomington, Ill., on April 21, 22 and 23. Dr. A. E. Braley, New York City, N. Y., will give several lectures on "Diseases of the Eye." Dr. Louis Clerf, Philadelphia, Pa., will discuss "Laryngeal Diseases." Dr. Louis Ostrom, of Rock Island, Ill., and Dr. H. Middleton, of Alton, Ill., will also give papers. For further information address the Secretary, Dr. Philip R. McGrath, 843 Jefferson Building, Peoria, Ill.

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Dysonic Model 1.
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Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

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Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

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Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

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Televox Model E.

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Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

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